PREVALENCE OF CRANIOFACIAL PAIN AS ONE OF THE SYMPTOMS OF MYOCARDIAL ISCHAEMIA AND MYOCARDIAL INFARCTION

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in partial fulfillment of the requirements

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“Hope deferred maketh the heart sick, but a longing fulfilled is a tree of life”

Every good and perfect gift is from Above. This good thing wouldn’t have been possible if Thy grace wasn’t sufficient. A big Thank you Lord!

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My Dynasty My priceless family

My parents, My sister DR.A.christina, Mr.V.Jasper my brother in law , Mr.V.Hudson my cousin and My husband Mr.M .Sam Titus and my daughter Amanda for their prayers and support. Without them it would have been impossible.

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TRIPARTITE AGREEMENT

This agreement herein after the "Agreement" is entered into on this day of 21 / 12/ 2016 between the Best Dental Science College represented by its Principal having address at Best Dental Science College, Madurai-625104.(Hereafter referred to as "the college")

And

MRS.DR.CH.UMA REDDY.M.D.S., aged 40 years working as Professor and Head of the Department at the college. , having residence address at No 9 staff quarters,ultra's Best Dental Science College,Madurai-625104.Here in after referred to as the Principal Investigator

And

DR.A.STEFFINA LYDIA JASCINTH aged 27years studying as postgraduate student in the Department of Oral Medicine and Radiology in Best dental science college. Herein after referred to as the PG/Research student and co-investigator.

Where as PG/Research student as part of her curriculum undertakes to research "Prevalence of Craniofacial pain as one of the symptoms of Myocardial Ischaemia and Myocardial Infarction" for which purpose PG/Principal Investigator shall act as Principal Investigator and the college shall provide requisite infrastructure based on availability and also provide facility to the PG/Research student as to the extent possible as a Co-investigator.

Whereas the parties ,by this agreement have mutually agreed to the various issues including in particular the copyright and confidentiality issues that arise in this regard.

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7. The Principal Investigator shall suitably guide the student Research right from selection of the Research Topic and Area till its completion. However the selection and conduct of research ,topic and area research by the student researcher under guidance from the principal investigator shall be subject to the prior approval ,recommendations and comments of the Ethical Committee of the college constituted for this purpose

8. It is agreed that as regards other aspects not covered under this agreement, but which pertain to the research undertaken by the student Researcher, under guidance from the Principal Investigator, the decision of the college shall be binding and final.

9. If any dispute arises as to the matters related or connected to this agreement herein it shall be referred to arbitration in accordance with the provisions of the arbitration and conciliation Act ,1996.In witness whereof the parties herein above mentioned have on this the day month and year herein above mentioned set their hands to this agreement in the presence of the following two witness

Principal

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ABSTRACT

Aim

To determine the Prevalence of craniofacial pain in patients with Myocardial Ischaemia and Myocardial Infarction .

Materials and Methods

A total of 1000 consecutive patients with confirmed myocardial ischemia (882 males, 118 females, mean age 53.9 years) were studied. Demographic details, health history, risk factors, prodromal symptoms, electrocardiogram (ECG) findings, and pain characteristics were assessed. Chi square and 'p' values were calculated . Student's 't' test was used to test the significance of difference between quantitative variables and Yate's and Fisher's chi square tests for qualitative variables. A 'p' value less than 0.05 denotes significant relationship.

Results

Craniofacial pain was more prevalent in Myocardial Infarction in the age group of 51-60 yrs , in females and those who had hypertension. craniofacial pain was associated with profuse sweating and those who had the habit of smoking.

Conclusion

Craniofacial pain is multifactorial, but cardiac origin should be considered when pain is burning, stabbing in nature radiating bilaterally to the jaws associated with symptoms such as dyspnoea , diaphoresis ,palpitations and giddiness. This needs to be considered as cardiac emergency.

Key words: Craniofacial Pain, Myocardial Infarction, Myocardial Ischaemia,Electrocardiogram

ABBREVIATION	ACRONYM
LV	LEFT VENTRICLE
NSTEMI	NON ST ELEVATION MYOCARDIAL INFARCTION
IASP	INTERNATIONAL ASSOCIATION SOCIETY OF PAIN
AMI	ACUTE MYOCARDIAL INFARCTION
ECG	ELECTROCARDIOGRAM
EMS	EMERGENCY MEDICAL SERVICES
CFP	CRANIOFACIAL PAIN
CNS	CENTRALNERVOUS SYSTEM
OPG	ORTHOPANTOMOGRAM
TMJ	TEMPOROMANDIBULAR JOINT
ACS	ACUTE CORONARY SYNDROME
MI	MYOCARDIAL INFARCTION
CVD	CARDIOVASCULAR DISEASE
HDL	HIGH DENSITY LIPOPROTEIN
NO	NITRIC OXIDE
ROS	REACTIVE OXYGEN SPECIES
PAI	PLASMINOGEN ACTIVATOR INHIBITOR
NOS	NITROGEN OXYGEN SPECIES
METC	MITOCHONDRIAL ELECTRON TRANSPORT CHAIN
NADPH	NICOTINAMIDE ADENINE DINUCLEOTIDE PHOSPHATE
CS	CIGARETTE SMOKING
CAD	CORONARY ARTERY DISEASE
TG	TRIGEMINAL NERVE
CGRP	CALCITONIN GENE RELATED PEPTIDE
GPCR	G PROTEIN COUPLED RECEPTOR

TSN	TRIGEMINAL SPINAL NUCLEUS
NGF	NERVE GROWTH FACTOR
RAGE	RECEPTOR FOR ADVANCED GLYCOSYLATION
CHD	CORONARY HEART DISEASE

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“Pain is far more than a simple sensation; pain is an experience.”

IASP defines Pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." Pain is one of the most feared sensation in medicine and dentistry. It is complex physical, psychological and social experience. Craniofacial pain is frequently associated with misdiagnosis and unnecessary therapy directed to the pain location instead of its origin.[1]

Pain creates an emergency for the patient and therefore becomes a prime motivating cause for seeking aid. When the suffering occurs in and around the mouth, the dental practitioner is usually the first to be called. When it occurs around the ears, face, head or neck a physician may be consulted. The actual location of the symptom may well determine who the patient sees first. Yet the location of the pain can mislead both patient and practitioner as to its true source and significance. Referred pain, the experience of pain in a location which is distant from its source, is a common phenomenon and can present a considerable challenge to clinicians.[2] Failure to diagnose the true cause of referred pain can lead to treatment delay and unnecessary therapy directed to the pain location instead of its real origin and, in the case of referred pain prior to a myocardial infarction, could be life threatening.[3]

In particular, patients with acute myocardial infarction (AMI) who do not experience chest pain run a very high risk of misdiagnosis and death. Cardiac pain most commonly radiates to the left arm, shoulder, neck, face, and in some instance radiates to tooth and its associated structures which is completely unrelated. Cardiac induced referred pain to the craniofacial region may drive a referral to a dentist and potentially miss the diagnosis of a life threatening cardiac condition to any dental source of origin.[4]

Even though coronary disease is the leading cause of death in developed countries, clinicians and the general public are unaware of certain atypical presentations of Myocardial Infarction which often delay going to the hospital, with 40% waiting longer than six hours past symptom onset.[5]

This means that the patient has potentially experienced an expanded myocardial injury during this period. EMS providers must attempt to compensate for delay by quickly recognizing the AMI, treating it appropriately and transporting the patient to the appropriate facility. [5]

The pain descriptor, pressure and burning sensation were associated with pain from cardiac origin while throbbing and aching indicated an odontogenic cause.[2]

Heterotopic pain within the craniofacial area can be generated by several conditions; odontogenic, myofascial and temporomandibular joint pains are well-documented examples of pain conditions that often present with a referred pain pattern. The characteristic symptom of Ischaemic heart disease is chest pain, which may radiate to the shoulders, arms and neck. However, cardiac pain may extend to the jaws and cause toothache. It has been stated that craniofacial pain was the sole symptom of Myocardial Ischaemia in 6% of patients. When craniofacial pain is the sole symptom of myocardial ischaemia, failure to recognize its cardiac source can endanger the patient.[6]

In a prospective multicenter study, it was found that the pain in the craniofacial structures was the only complaint during Myocardial Ischaemia.[7]

In Ischaemic cardiomyopathy, the painful stimulus is triggered by oxygen deprivation in the coronary arteries; the patient may then suffer angina, if no cellular necrosis of the tissue is present, or may otherwise cause a heart attack. Consequently, the symptoms of the supposed odontalgia will decrease with vasodilators such as nitro glycerine, or with the revascularization of the damaged area.[8]

The methods for detecting Ischaemic cardiopathy include three basic pillars: clinical exploration, an electrocardiogram and the markers of myocardial damage. In many cases, the clinical pain which is different from odontogenic pain will be accompanied by symptoms such as sweating, vomiting and dizziness.[9]

The differential diagnosis of pain of odontogenic origin (dental and periodontal) must always be taken into account with pain of non-odontogenic origin (muscle pain, psychogenic, neuronal, cardiac, sinus and neurovascular) in order to avoid diagnostic errors in the dental practice as well as performing unnecessary treatments. **Orofacial pain of cardiac origin is a bilateral pain, mainly located in the mandible and throat.[6]**

It can radiate to other craniofacial structures and also to more common areas such as the arms, shoulders and chest. It is paroxysmal and severe, but less intense than toothache and, additionally, less intense at more atypical areas of location of cardiac pain. [6]

A common site for the referred pain in patients with Myocardial Ischaemia who aren't experiencing chest pain is the craniofacial area (38–60%). For 6% of patients, this was the only complaint. The most common location is the upper throat (82%). This is followed by the mandible (45%) or the left temporomandibular joint/ear (18%). [3]

The possibility of referred craniofacial pain being the sole symptom of Myocardial ischaemia is low because of lack of awareness that craniofacial pain could be the only symptom of Myocardial ischaemia. Temporomandibular joint and jaw pain induced by Myocardial Ischaemia tends to occur bilaterally as opposed to referred pain of dental origin. [2]

Furthermore, patients who experience an AMI with atypical symptoms are more likely to be misdiagnosed and discharged from emergency departments than patients with typical presentations. Early treatment of acute coronary disease plays a critical role in improving outcome.[3]

In fact, reperfusion therapy within the early phase of an AMI has been shown to significantly reduce the inhospital mortality of patients and each 30-minute delay was associated with an increased relative risk of 1-year mortality.[3]

Similarly in dental practice, approximately 1% of medical emergencies result in patient death and are mostly associated with cardiac failure .

AIMS AND OBJECTIVES

- To determine the Prevalence of the craniofacial pain in patients with Myocardial Ischaemia and Myocardial Infarction
- To evaluate the most common location of pain in the craniofacial region due to cardiac origin.
- To determine whether there is radiation of pain of cardiac origin to other sites along with craniofacial pain
- To evaluate if there is any associated symptoms along with craniofacial pain of cardiac origin
- To determine any differences in quality and distribution of craniofacial pain of cardiac origin from dental origin
- To evaluate any association between craniofacial pain of cardiac origin and risk factors , age and sex .
- To compare whether craniofacial pain is more prevalent in Myocardial Ischaemia or Myocardial Infarction.

Review of Literature

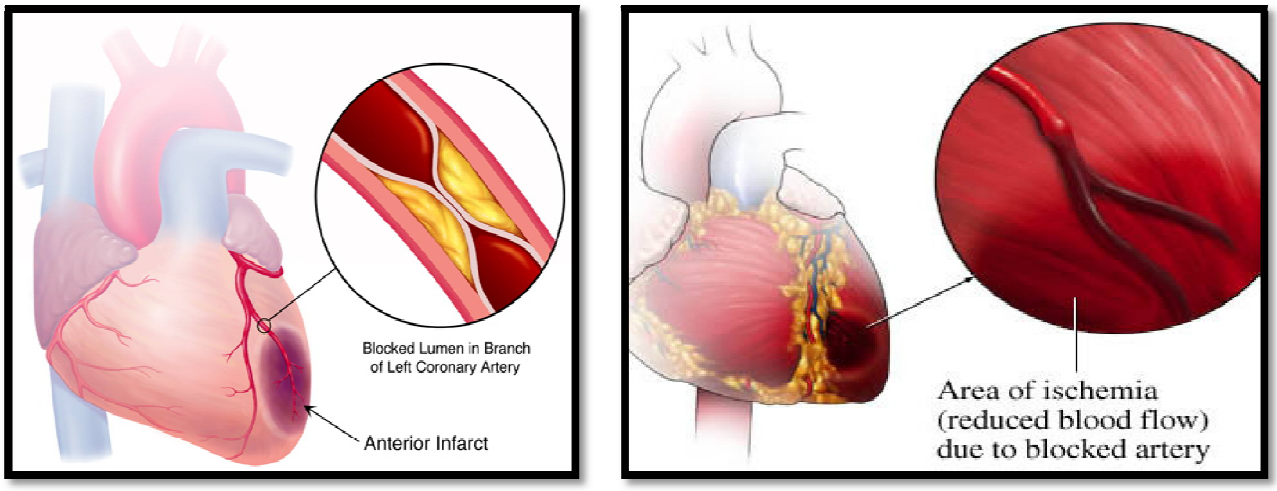


Fig:1 Difference between Myocardial Ischaemia and Myocardial Infarction

Courtesy:http://www.differencein.com/distinction-between-ischemia-and-infarction[10]

AMI

Acute myocardial infarction (AMI) is often overlooked or misdiagnosed. Those who present atypically often have "anginal equivalent complaints" that providers should be aware of. These include epigastric discomfort, general weakness and other nonspecific complaints. In fact, only 25% of the elderly present with the classic triad of chest pain, ECG abnormalities and serum markers corresponding to their MIs. Nondiagnostic ECG findings are present for 50–75% of elderly patients experiencing MI. Further, certain medications (like digoxin) can alter the ST segment such that a patient won't have ST elevation.[11]

The sudden rupture of an atherosclerotic plaque, with ensuing intracoronary thrombus formation that acutely reduces coronary blood flow, causes the acute coronary syndromes (ACSs). This results in myocardial ischaemia and subsequent infarction if there is a prolonged and severe reduction in blood flow.[11]

Acute coronary syndromes represent a continuous spectrum of disease ranging from unstable angina (USA) to non-ST-elevation MI (NSTEMI) to acute ST-elevation MI (STEMI). If the intraluminal thrombus following acute plaque rupture is not completely occlusive, the corresponding clinical presentation is that of USA or NSTEMI. There is a sudden change in anginal pattern relating to the frequency or duration of the symptoms. [11]

In some cases, the patient may present with symptoms at rest. With a greater degree of obstruction of the epicardial coronary arterial lumen, a non-Q-wave MI (NQWMI) may develop. This presents with prolonged symptoms of resting ischaemia, typically without ST-segment elevation or the development of pathologic Q waves. Electrocardiography in a NQWMI patient may show resting ST-segment depression or deep symmetric T-wave inversions, consistent with severe ischaemia. If a large epicardial coronary artery becomes obstructed for a relatively long duration of time, a larger myocardial infarct results, and the electrocardiographic findings will be STEMI and, without prompt restoration of blood flow, the subsequent development of pathologic Q waves.[11]

Diagnosis of Acute Coronary Syndromes

The electrocardiogram is the most important diagnostic tool to risk-stratify the patient and to make decisions regarding treatment. A normal ECG does not exclude the presence of acute MI (AMI). If a STEMI is located at the posterior wall of the left ventricle (LV), it will typically not be well represented on the standard 12-lead ECG. [11]

Resting ST-segment depression or T-wave inversions in the distribution of an epicardial coronary artery often accompanies USA or NQWMI; however, ST-segment elevation is the hallmark of an acute STEMI.

Patients presenting with a history suggestive of an AMI who have a left bundle branch block pattern on the 12-lead ECG are usually treated as if they had STEMI, given the difficulty of interpreting the ECG when this conduction delay is present.[11] Neuropathies of the autonomic nervous system, cortical failure, damage to cardiac sensory nerves caused by heart disease, increased pain threshold and any comorbidities, including dementia, all contribute to atypical presentations. The most common atypical presentation of the silent AMI is dyspnoea. Other likely presentations include general weakness, fatigue, cold sweats or dizziness. Providers also shouldn't dismiss a syncopal episode as the primary symptom of an MI, as this occurs in 3% of elderly patients and is correlated with high mortality.[11]

Tzukert reported Orofacial pain of cardiac origin in 1981.[2]

Anika Isberg correlated the signs and symptoms of craniofacial pain as a result of cardiac ischaemia and also stated that one in three patients with craniofacial pain with no chest pain during Myocardial ischaemia may develop acute myocardial infarction.[2]

Chuckwuemeka 1999 reported a case of unilateral facial pain that occurred in relation to a large pericardial effusion and which resolved on drainage of the effusion.[12]

According to **Everts et al 1996** Patients in whom AMI developed localized their pain to an extent similar to those without AMI in seven of nine chest areas. However, patients with AMI reported pain in the upper right square of the chest more frequently and in the middle left square of the chest less frequently than did patients without AMI. Pain in both the right and left arms was more frequently reported by patients who had AMI. [13]

Among patients with AMI, women reported pain in the neck and in the back more frequently than did men. Compared with elderly patients, younger patients reported pain more frequently in the left arm , right arm , and neck. Both patients were finally diagnosed as suffering from ischaemic cardiomyopathy, with referred pain to the face. Pain quality, intensity and gender characteristics were assessed for referred craniofacial pain from dental as well as of cardiac origin**.** No gender differences were found. This clinical presentation can be expected in one in fifteen patients, more prevalent in women than in men. [13]

Oliveria Franco et al reported A case of a 65-year-old female patient with facial pain, so she sought care from her general dental practitioner for evaluation of a suspected temporomandibular disorder after repeated visits then she went to the emergency department due to excruciating facial pain associated with exertion.[14]

In these settings, dentists may contribute to the diagnosis of ischaemic heart disease and refer patients for cardiological evaluation.[14]

According to **Gupta et al 2002** out of 721 myocardial Infarction cases 53% of patients presented with chest pain. The frequency of other complaints were shortness of breath, 17% (121); cardiac arrest, 7% (50); dizziness/weakness/syncope, 4% (32); abdominal pain, 2% (14); and other, 17% (124). The risk of a presentation without chest pain in a patient with AMI increased with age. The characteristic with the highest risk for a presentation without chest pain in patients with AMI was age older than 84 years old . Women were more likely than men to present without chest pain.[15]

According to **Franco et al in 2006**, A 50-year-old female patient was referred to a dentist for evaluation of a suspected temporomandibular disorder after repeated visits to medical emergency departments due to excruciating facial and left temporal pain associated with exertion. [16]

The pain would start in the chest and radiate to the neck, face and left temporal region.A diagnosis of temporomandibular disorder was made. However, she was referred for cardiological evaluation, since her pain was starting in the chest and because she had a past medical history of surgical treatment for coronary artery disease. A diagnosis of angina pectoris was made, the therapeutic regimen was optimized and her angina was brought under control.[16]

A multicentric study conducted by **Kreiner et al. in 2007** The primary objectives of this first study were determining the prevalence of orofacial pain on a sample of 186 patients presenting ischemic heart disease as well as describing the location and irradiation of pain. However, were not described other characteristics important to perform a correct and differential clinical diagnostic, such as kind, frequency, intensity, the triggering factors and how to alleviate it. [8]

During **2010, Kreiner et al**. published another study aiming to differentiate the kind and intensity of toothache in comparison to orofacial pain of cardiac origin. Toothache is described as pulsatile and sharp whereas pain of cardiac origin is described as oppressive and burning. Additionally must be noted that the intensity of pain was higher in patients with toothache than in those with pain of cardiac origin. [17]

At intraindividual level the craniofacial pain of cardiac origin is less intense than toothache. However, the intensity increases in locations closer to the heart.[17]

Pain originating in the heart in craniofacial structures is usually bilateral, whereas odontogenic pain is always unilateral. The most frequent location described for craniofacial structures is in the throat and mandible . [17]

However, in the literature, we find other orofacial locations where the cardiac pain originates: neck, maxilla, zygomatic arches, head, temporomandibular joint, ears and teeth . Due to its location, pain of cardiac origin is considered unusual. [17]

However, studies such as those conducted by Kreiner et al. show that for 1 out of every 15 patients who present cardiac ischaemia, it manifests in the craniofacial structures. Considering that ischaemic cardiopathy is one of the main causes of death among the adult population, there is clearly a clinical underestimate considered to be atypical clinical features, and therefore, this data is significant .[17]

Pain of cardiac origin manifesting in the orofacial area may irradiate to other craniofacial structures (throat, neck, temporal area, head, infraorbital region, maxilla) or to the thorax region (thorax, shoulders, arms). Odontogenic pain (pulpal or periodontal) can be reflected in structures such as the ears and the temporal area. However, pain of dental origin never refers to the typical areas of precordial pain, such as the thorax, arms and shoulders. [6]

According to Kreiner et al. 32% of the patients presented concomitant craniofacial pain in other regions and only 6% presented craniofacial pain as the only symptom during the ischaemic episode. Craniofacial pain was predominantly present in females and was the main symptom in both sexes, without the presence of chest pain.[17]

However, it is necessary to conduct broader studies and with a larger sample of patients, in order to determine the characteristics of the orofacial pain of cardiac origin, to avoid unnecessary dental treatments such as dental extractions and non-indicated temporomandibular dysfunction therapies, and to not delay the correct diagnosis of heart disease .[17]

The only comparative study between the quality of dental pain and heart pain, emphasizes that dental pain presents characteristics pulsating and stinging compared with cardiac pain described as burning and oppressive. The onset of the pain is usually spontaneous and can be triggered after performing physical exercise. It usually remains unchanged by movement or oral stimuli and is usually alleviated with adequate cardiac treatment.[6]

According to **Dalband et al 2011** a 48-year-old man with a chief complaint of severe bilateral pain in the temporomandibular joint was referred for evaluation of a suspected temporomandibular disorder. The patient was referred for cardiological evaluation (exercise test, electrocardiography, laboratory tests and coronary angiography) and was diagnosed with angina pectoris. The patient had no previous history of heart disease or chest pain. In conclusion, awareness of this symptomatology can be useful for diagnosis of coronary insufficiency and timely treatment. Therefore, cardiac disease should be considered in the differential diagnosis of orofacial pain.[18]

In a study conducted by **Danesh et al 2012** a prospective study of 248 consecutive patients (aged 26 to 88 years) hospitalized with confirmed cardiac ischaemic periods. Digital OPG radiographs were obtained from all patients for radiographic examination of the jaws and dentition. Patients underwent clinical and radiographic examinations, and symptoms were evaluated in detail to determine the prevalence and distribution pattern of craniofacial pain of cardiac origin.[19]

Craniofacial pain was the sole symptom of cardiac ischaemia in 13 patients (5.2%); two developed acute myocardial infarction (AMI). Pain in the craniofacial region, chest, shoulders and arms was experienced by 72 patients. The most frequently affected region was the left mandible.[19]

In the absence of chest pain, patients most frequently experienced pain in craniofacial structures. Incidence of craniofacial pain was significantly higher in females than males .[19]

According to **Lopez et al 2012** The sample of that study included thirty patients when doing a treadmill exercise test.Eleven of the 30 patients included in this study presented craniofacial pain before or during the cardiac seizure. The location of the pain was bilateral, non-irradiated at the mandible in all cases. The intensity of the pain was from slight to severe. The frequency of the appearance of the pain was paroxysmal in 8 cases and constant in three cases, and the duration was from a few hours to a maximum of 14 days. [6]

The cardiac pain in craniofacial structures is usually bilateral, compared to odontogenic pain which is always unilateral.[6]

The pain of cardiac origin is considered atypical because of its location, but about the 10 % of the cases, the cardiac ischaemia has its primary manifestation in orofacial structures. Eleven patients referred a bilateral non-irradiated mandibular pain, with intensity from slight to severe, and with a paroxysmal frequency in eight cases and a constant frequency in three cases. Just one patient referred pain during the treadmill exercise test. In all cases the pain disappeared after the cardiac surgery or the administration of vasodilators.[6]

According to **Muhammed et al 2013**, A total number of 331 patients with AMI were included in the study. Mean age was 54.99±11.25 years with minimum age 20 years and maximum age 90 years. It included 264(79.8%) male and 67(20.2%) female patients with male to female ratio of 3.9:1. Out of these 331 patients 308 (93.1%) patients reported chest pain as the presenting complaint. Remaining 23(6.9%) presented with clinical features other than chest pain. There were 127(38.4%) patients with pre-cordial chest pain, 115(34.7%) had retrosternal chest pain, 58(17.5%) were having epigastric pain.[20]

Severe chest pain was seen in 281(84.9%) patients while 26(7.9%) had only mild chest discomfort. Radiation of the pain to shoulder, neck and jaw was seen in 75 (22.7%) patients. In 42(12.7%) patients, pain radiated to both sides of chest.[20]

Another 55(16.6%) patients had pain radiation to chest, shoulder, upper arm and ulnar side of left forearm. Chest pain radiation to interscapular region along with both sides of chest was present in 10(3.0%) patients. In 11(3.3%) patients pain radiated only to left side of chest. Pain persisting for >20 minutes was reported by 298 (90%) patients while only 10(3.1%) had pain persisting for <20 minutes.[20]

If myocardial ischaemia is intense or prolonged, it can lead to cardiac cell death and release of biomarkers of myocardial infarction (MI) such as troponin *I* and troponin *T*, which can be detected by specific laboratory testing .

According to clinical manifestations, electrocardiography (ECG) and serum levels of biomarkers, ACS can be divided in two categories including unstable angina (new or changing symptoms in a crescendo pattern that can also occur at rest, contrary to stable angina) and MI .

As outlined by **Okeson et al 2015**, the orofacial pain is classified into physical (Axis 1) and psychological (Axis 2) conditions.[9]

Physical conditions include disorders of the musculoskeletal structures for *e.g.* temporomandibular joint (TMJ), masticatory muscles and cervical spine; neuropathic pains which include episodic (*e.g.* trigeminal neuralgia) and continuous (*e.g.* peripheral/centralized mediated) pains and neurovascular disorders (*e.g.* migraine). Psychological conditions include mood and anxiety disorders.[9]

According to a systematic review, apart from psychogenic toothache, primary mechanisms that lead to sensation of non-odontogenic craniofacial pain (CFP) fall into two main categories: *i)* projected nerve pain including neuropathic toothache and neurovascular pain and *ii)* referred pain as a result of convergence and central sensitization such as myofacial pain, idiopathic toothache, sinus pain and cardiac pain referred to the craniofacial area . Dentists must to be aware of the possibility of ischaemic heart disease in patients who visit the clinic with complaints of toothache only; almost **40% of ischaemic heart disease patients experience facial pain during heart attacks with a significantly higher tendency in women while 15% experienced facial pain only .**[9]

Typically, general public recognize the cardiac pain as a localized pain in the sternal region and left side of the chest; however, it can also radiate to the neck, either arm, the shoulders, the stomach and the jaws .

The presence and intensity of pain is variable but is not associated with disease severity; both symptomatic and asymptomatic episodes can present with similar cardiac hemodynamic changes .[21]

Patients who experience CFP as the sole symptom of myocardial ischaemia are likely to seek dental treatment and thus the possibility of misdiagnosis is high.[21]

Until the date, the clinical link between CFP and myocardial ischaemia was limited to case reports . Toothache, mandibular pain, ear pain and headache were the most common reported pain locations. Misdiagnosis, mistreatment and delay in administration of appropriate therapy were common features in those reports.[21]

A cross sectional study was carried out to assess the prevalence of craniofacial pain among patients suffering from Myocardial Ischaemia and Myocardial Infarction in Government Rajaji Hospital Madurai.

- Ethical clearance

The synopsis of the proposed Research was prepared and submitted to the Institutional Review Board, Best Dental Science College and Hospital,Madurai and also Govt.Rajaji Hospital.Madurai for Ethical Approval.(Annexure I).After the review and scrutiny by the board members,approval was granted to conduct the research. The study was conducted after obtaining informed consent from the participants.(Annexure II)

- Detailed Case history format:

  The standard case history format was followed taking into consideration the history of Myocardial Ischaemia and Myocardial Infarction.(Annexure III)
- Quality of pain was assessed using Mcgill's pain questionnaire
- Intensity of pain was assessed using Visual Analog scale
- Intraoral examination was done using sterile mouth mirror and probe in1000 patients to rule out whether tooth pain or jaw pain is due to dental origin.

This study was carried out in a sample of 1000 patients who were diagnosed as Myocardial Ischaemia and Myocardial Infarction in the cardiology unit of Government Rajaji Hospital. Patients condition who were stable after initial medical line of treatment were examined detailed history of having experienced craniofacial pain including origin, site, nature, duration, distribution at the time of onset of Myocardial Ischaemia and Myocardial Infarction was noted.

Patient's dental examination was done using sterilised mouth mirror and probe to rule out pain of odontogenic origin thereby establishing craniofacial pain as one of the symptoms of Myocardial Ischaemia and Myocardial Infarction.

- Eligibility criteria

Inclusion Criteria

- Patients who are willing to participate in the study
- Males and Females who are suffering from Myocardial Ischaemia and Myocardial Infarction confirmed by ECG
- Age Group of 30-75
- Patients of Myocardial Ischaemia and Myocardial Infarction with other systemic disorders such as diabetes and Hypertension are taken into the study

Exclusion criteria

- Patients who are critically ill
- Patients who are not willing to participate in the study
- Patients below 30 yrs of age
- Patients with previous history of chronic orofacial pain

- Pilot study

A pilot study was done to know the feasibility of the study, to pretest the case history format and to calculate the sample size. Kappa statistics for intra examiner reliability was found to be 0.9.

- Content Validity

The case history format was verified by a panel of experts from the (Department of Oral Medicine and Radiology) and modified in accordance with their recommendations to ensure comprehensive ability. Data collected from these participants were used to make final refinements and levels of missing data was used as an indicator of inappropriate questions and that question was reframed accordingly.

Sample size calculation

Formula:$4pq/l^2$

sample size is 1000

- Study population

The study population included people of more than 30 yrs who visited Govt.Rajaji Hospital due to Myocardial Ischaemia and Myocardial Infarction confirmed by ECG as depicted in Fig2

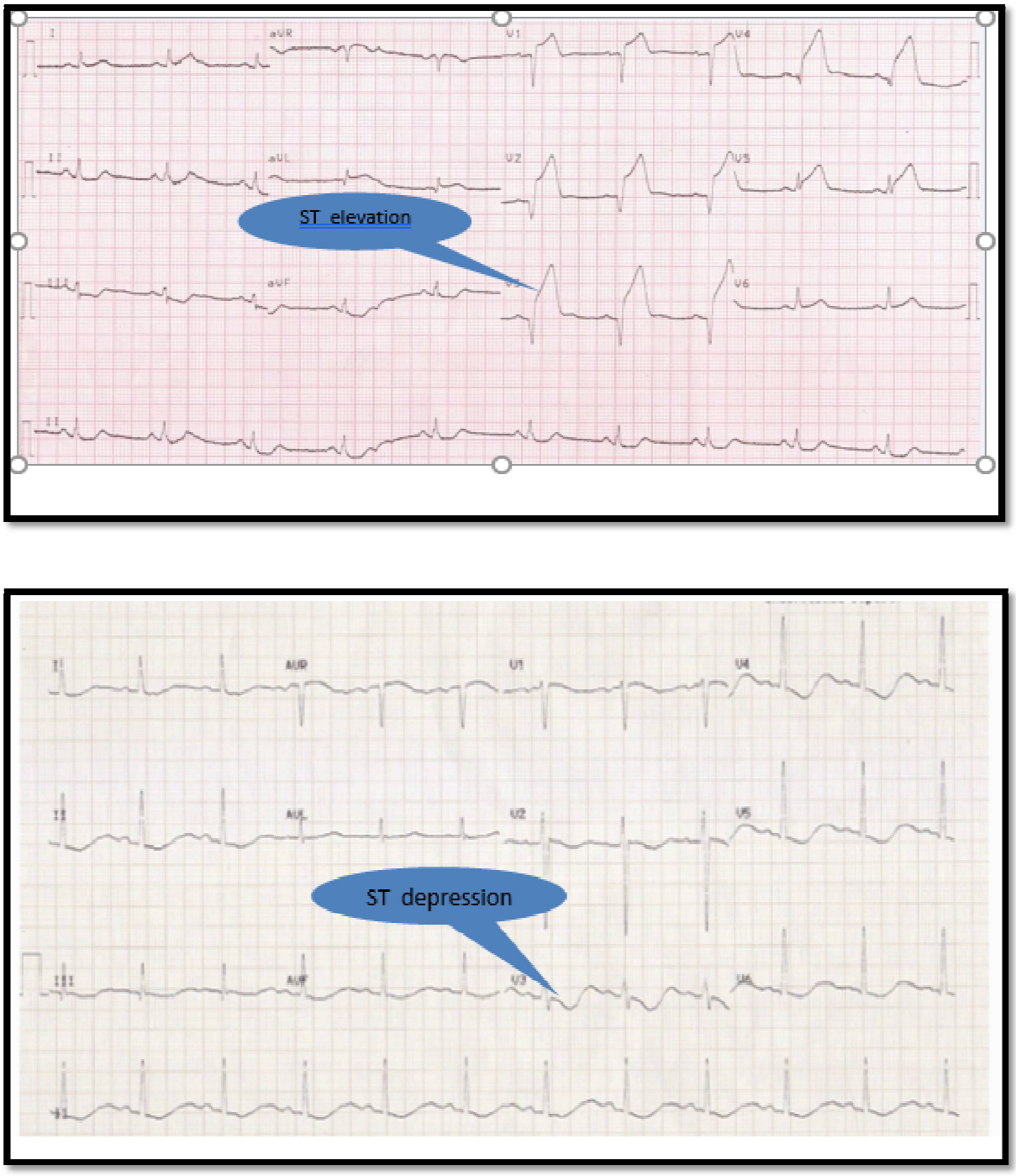


Fig:2 ECG shows ST Elevation and ST Depression

- Sampling Methodology

  Purposive sampling was done

- Collection of data

This was a cross sectional study in which case history format was developed and designed by considering the other previous studies. The participants were assured about the confidentiality of their personal information. The questions were asked to the study subjects in the local language or English depending on the convenience of the study subjects. They were informed to feel free and raise any questions to clarify their doubts.

- Statistical Analysis
- The information collected regarding all the selected cases were recorded in a Master Chart(Annexure IV).The entered data were checked for consistency. Dataset was subdivided and distributed meaningfully in individual tables. Data analysis was done with the help of computer using **SPSS statistical package- Version 17.**
- Using this software range, mean and standard deviations were calculated for quantitative variables like age.
- Frequencies and percentages were calculated for qualitative variables like sex, past medical history, personal history, associated symptoms, pain location etc.
- Chi square and 'p' values were calculated . Student's't' test was used to test the significance of difference between quantitative variables and Yate's and Fisher's chi square tests for qualitative variables. A 'p' value less than 0.05 denotes significant relationship.

FIG:3 STUDY CONDUCTED IN GOVT.RAJAJI HOSPITAL MADURAI

FIG:4 ECG MACHINE

FIG 5:PATIENT SUBJECTED TO ECG

FIG:6 STERILISED MOUTH MIRROR AND PROBE

FIG:7 RECORDING OF DETAILED CASE HISTORY

FIG:8 DENTAL EXAMINATION IN PATIENTS WITH MYOCARDIAL ISCHAEMIA AND MYOCARDIAL INFARCTION

Results obtained pertaining to Myocardial Infarction and Myocardial Ischaemia were categorized according to age, gender, associated symptoms, location of pain, type of pain, distribution of referred pain, comorbidities, oral status and associated habits.

Sample size of 1000 patients includes 796 patients of Myocardial Infarction and 204 patients of Myocardial Ischaemia which were diagnosed based on clinical features and ECG Findings as depicted in fig 9.

Fig 9:Pie diagram shows 796 (80%)patients had Myocardial Infarction and 204 (20%)patients had Myocardial Ischaemia. Prevalence of Myocardial Infarction was more than Myocardial Ischaemia

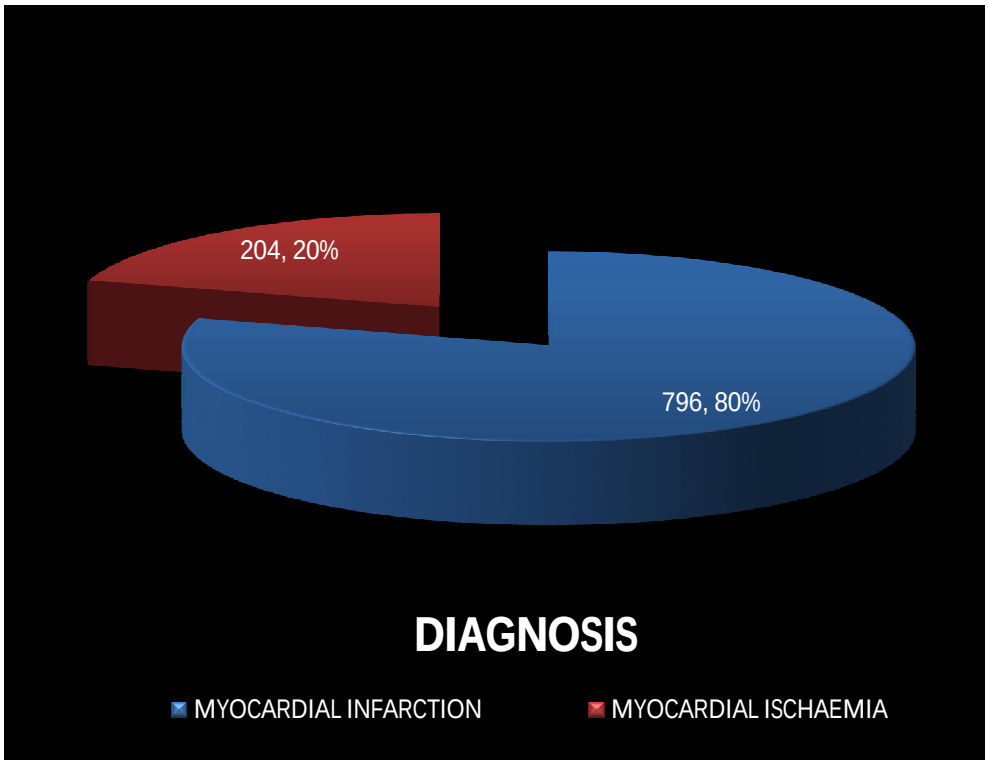


Fig:10 **Patients were categorized into Myocardial Ischaemia and Myocardial Infarction based on ECG findings**

Electrocardiogram was done in 1000 patients in the cardiology unit.

Pie diagram Shows ST Elevation was present in 80% of the population and ST Depression was present in 20% of the population

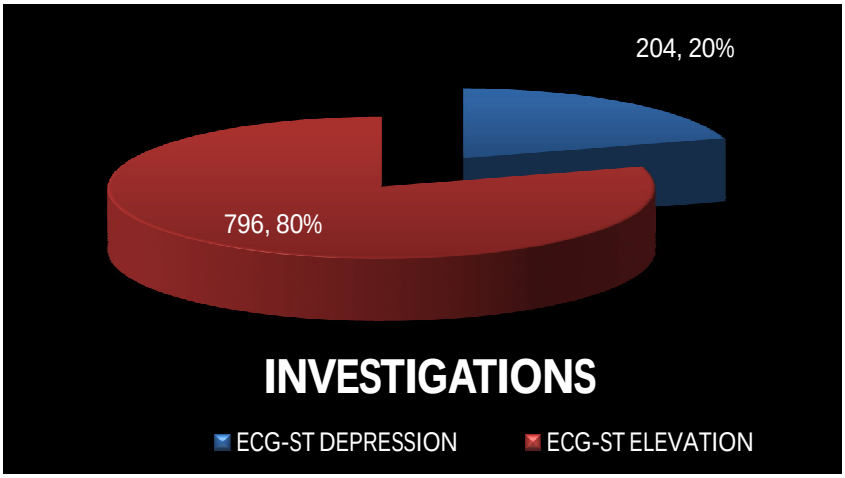


Fig:11 Represents Myocardial Ischaemia and Myocardial Infarction was more prevalent in the age group of 51-60yrs in bar diagram and the next group was in the age of 41-50 with mean age of 53.9 yrs.

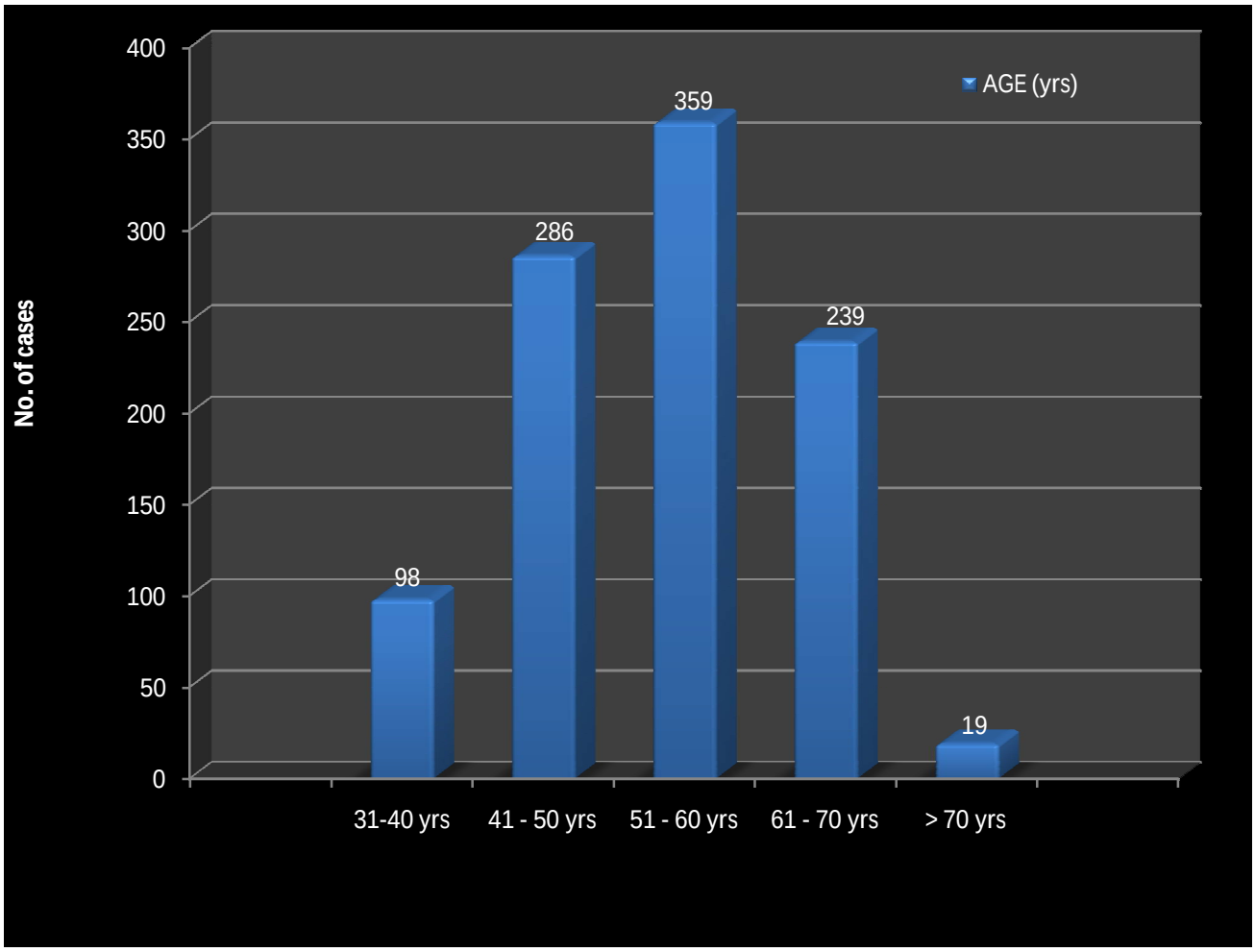


Fig:12 Myocardial Ischaemia and Myocardial Infarction was more prevalent in males (88%)compared with females 12%

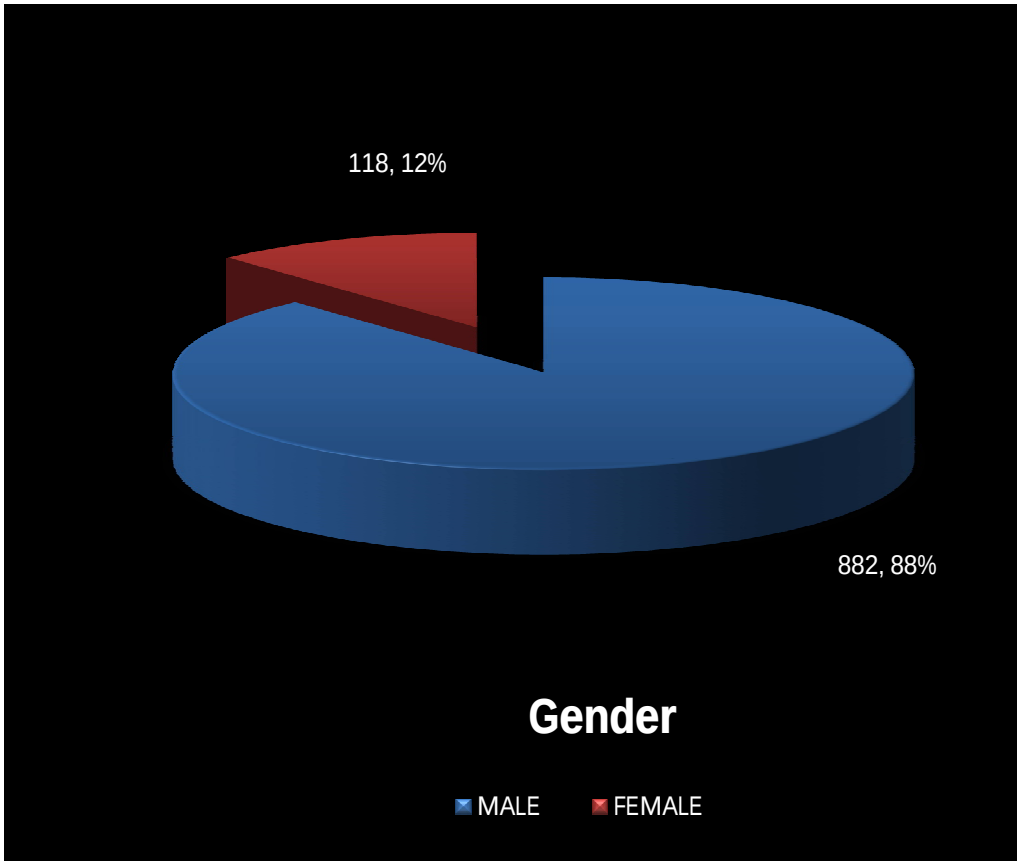


Fig:13

The most common associated symptom was profuse sweating in 16% of the population followed by dyspnoea in 14% of the population.5% of the population had vomiting and diarrhoea.3%of the population had giddiness and palpitation

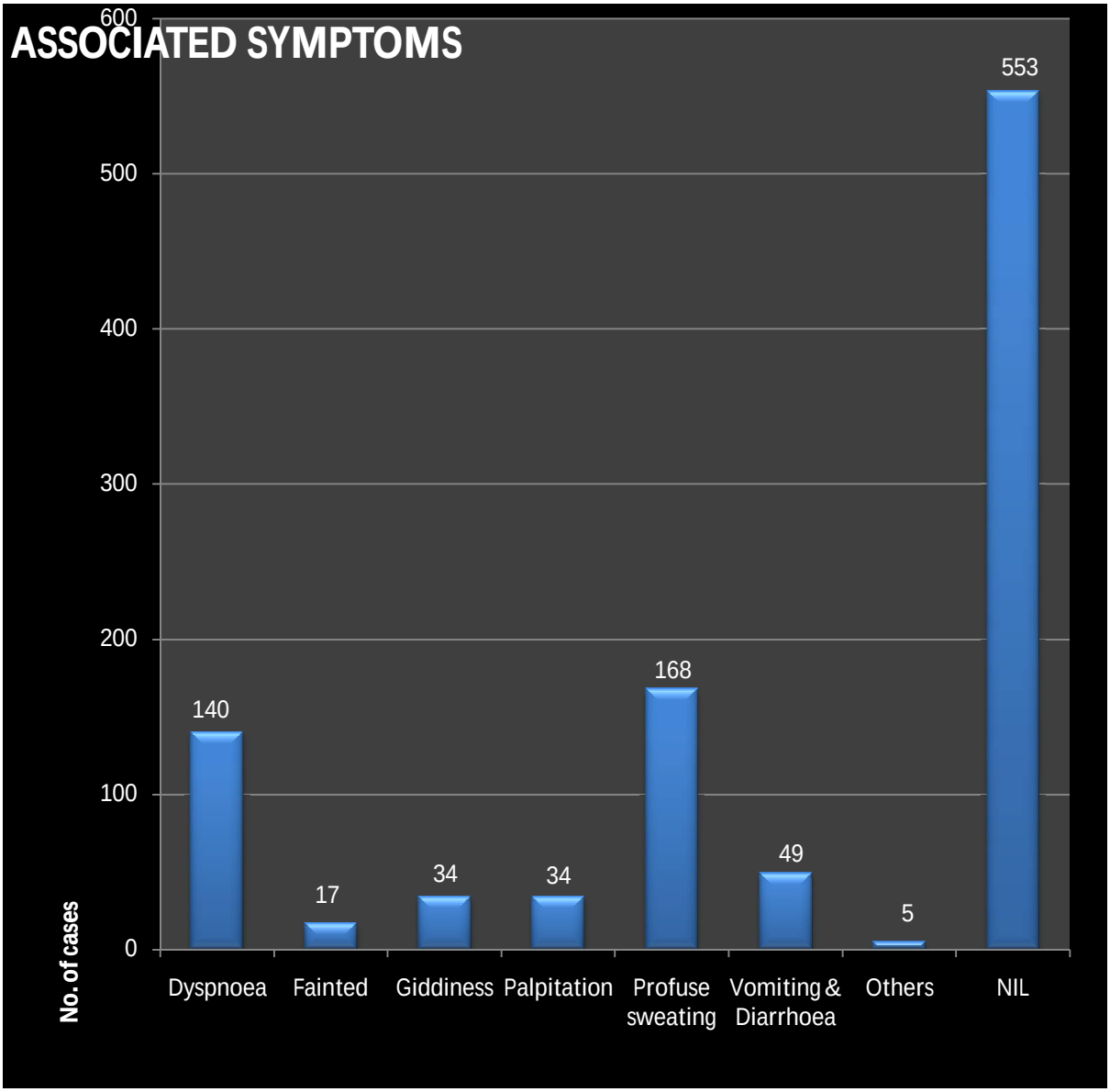
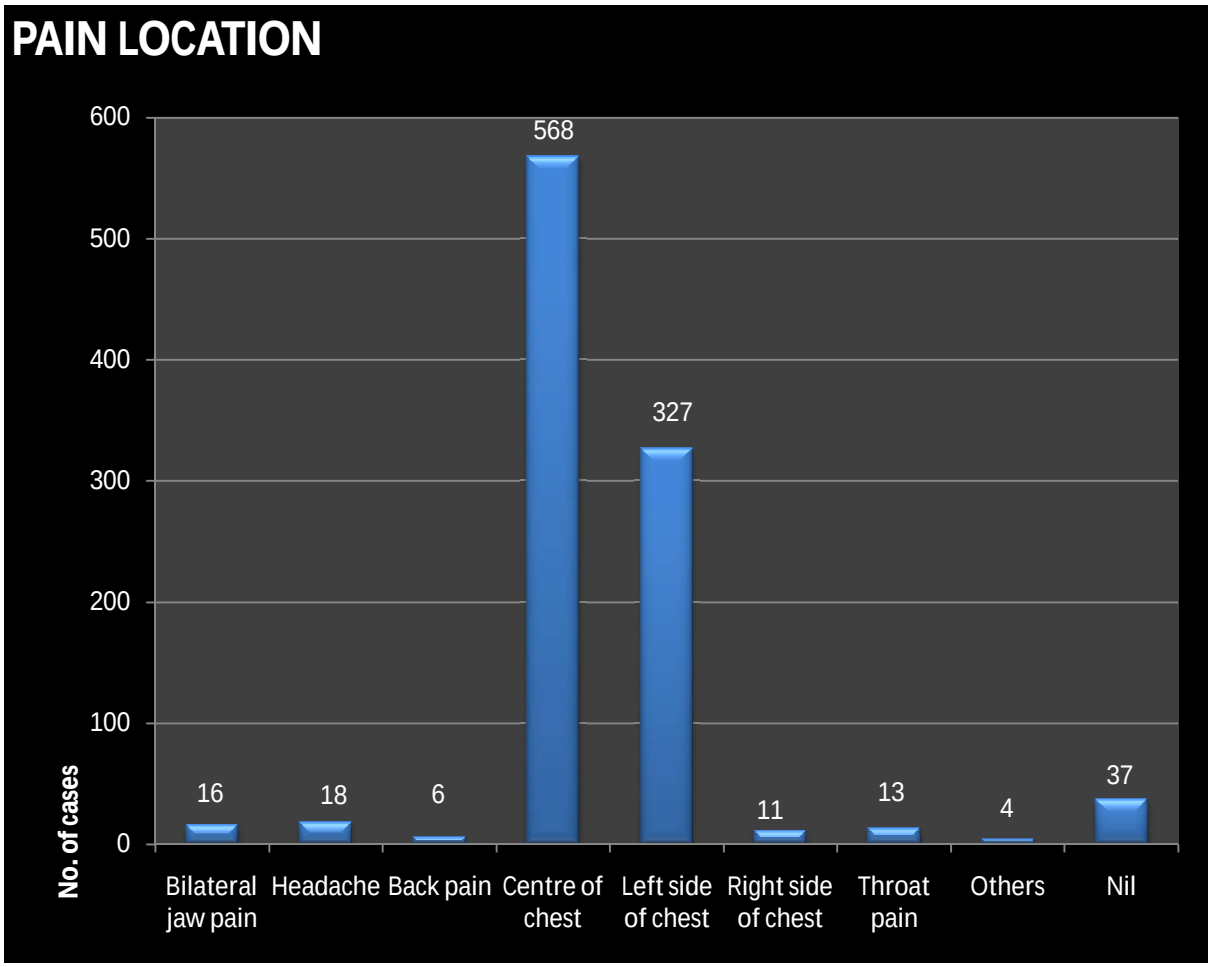


Fig:14 The most location of pain experienced by patients who had Myocardial Ischaemia and Myocardial Infarction was in centre of chest followed by left side of chest



- **Bilateral jaw pain and head ache was present is in 3%of the patients who experienced Myocardial Ischaemia and Myocardial Infarction**

Others include patients who experienced right and left hand pain and symptoms without any pain

Nil includes patients who had fainted

Fig:15 Bar diagram shows the most common type of pain in the patients who had Myocardial Ischaemia and Myocardial Infarction was of stabbing type(77%)

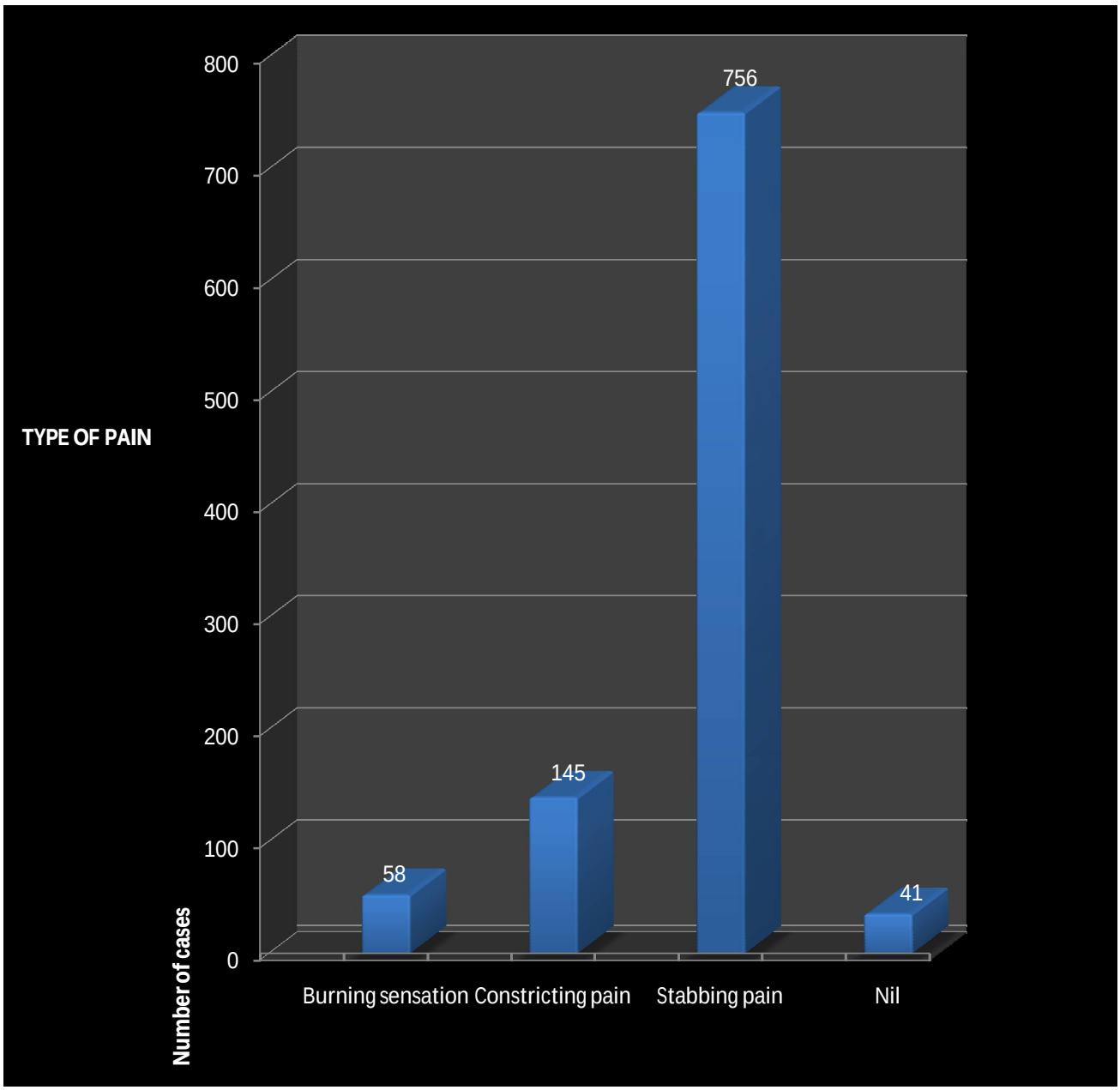


Fig:16 The most common distribution of pain was in left shoulder followed by back pain and right and left shoulder pain as depicted in bar diagram.Head ache was also experienced by the patients who had chest pain

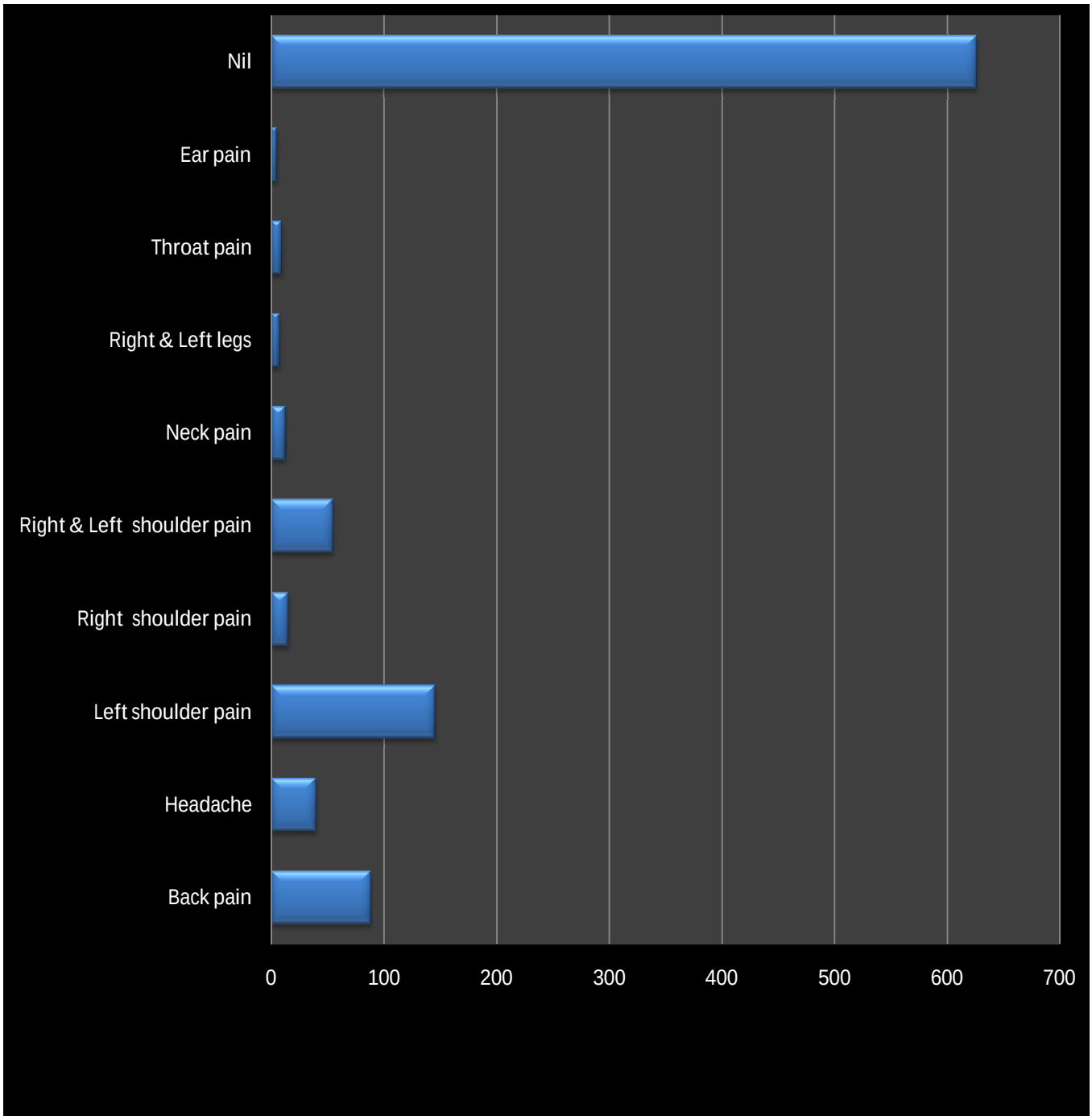
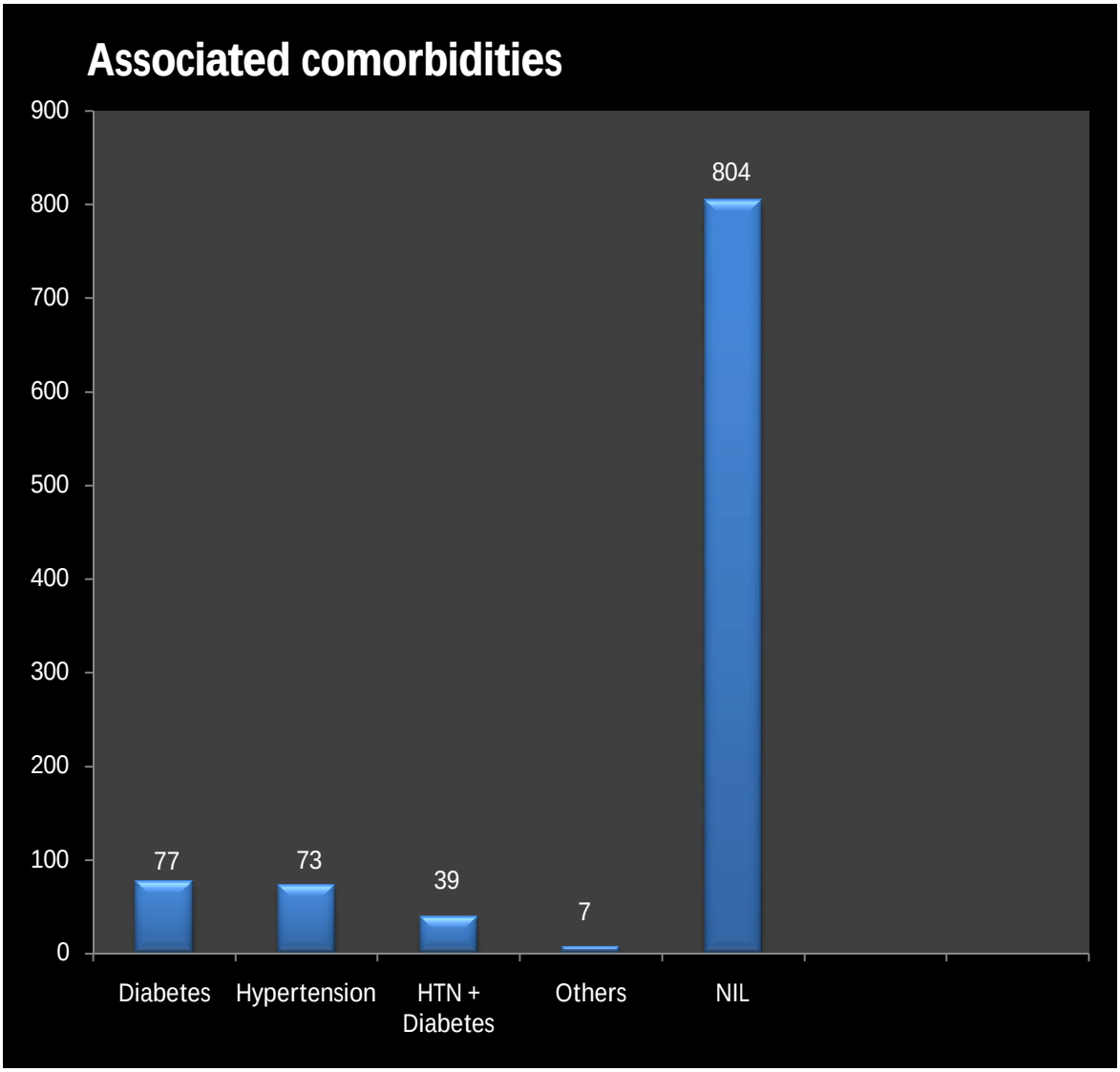


Fig:17 Bar diagram shows diabetes(8%) was the most common comorbidity in the patients who had Myocardial Ischaemia and Myocardial Infarction and hypertension was present in 7% of the population



Others include:Asthma,Hypothyroidism

Fig:18 shows the Oral status .The most common oral finding was Generalized chronic peridontitis followed by dental caries as depicted in the bar diagram. Attrition, edentulousness ,smoker's palate and smoker's melanosis was also present

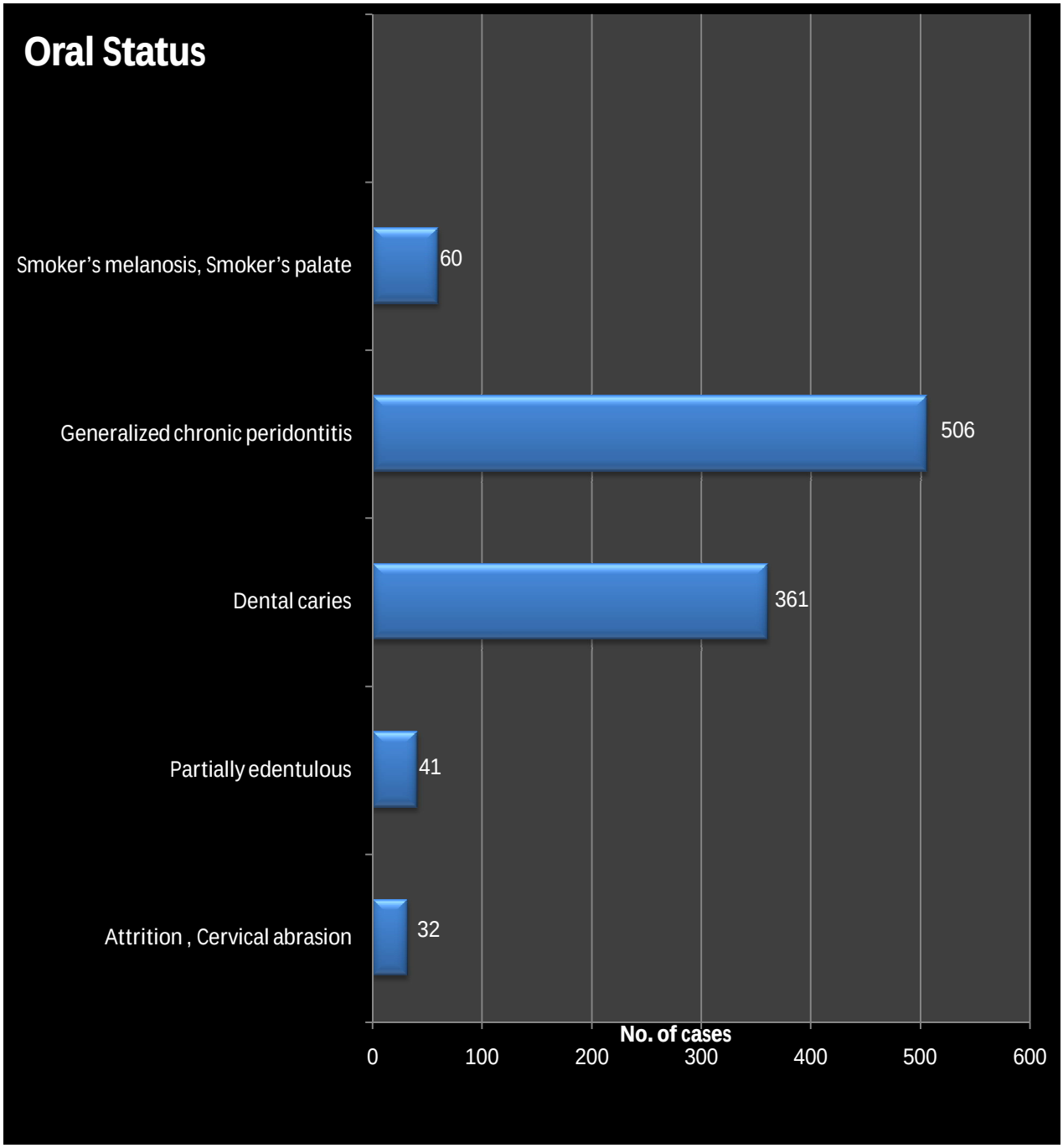
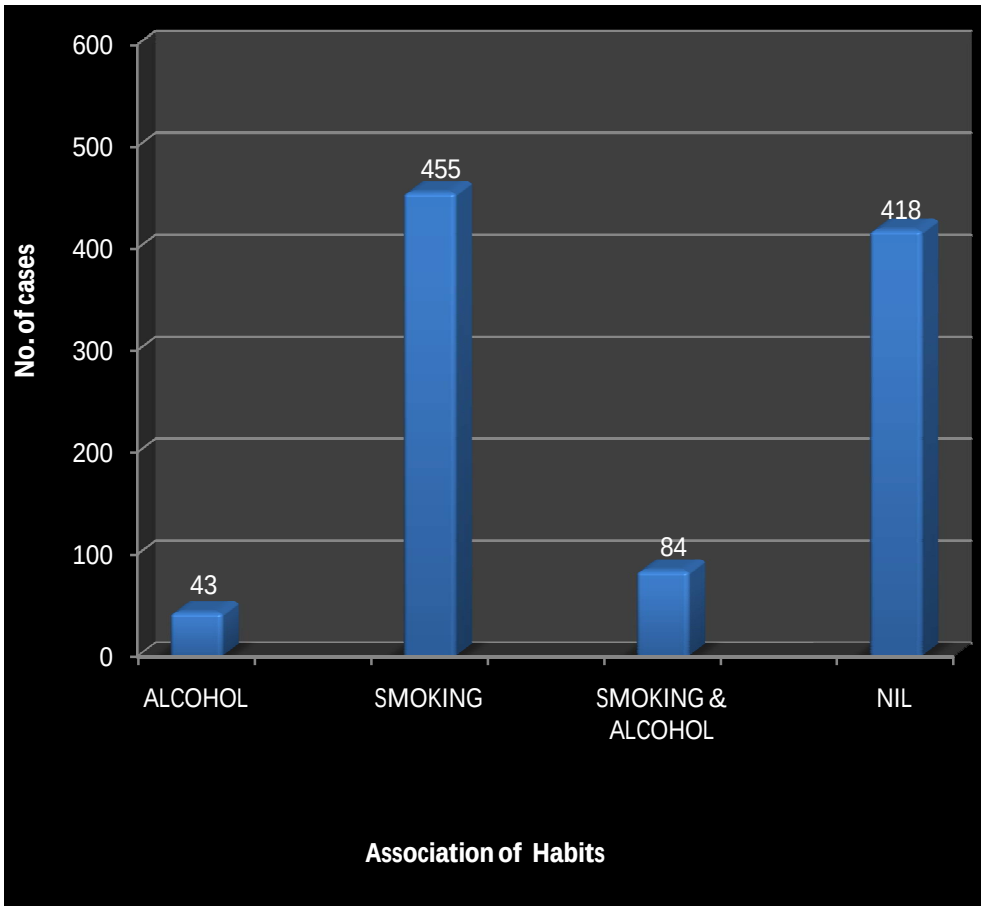


Fig:19 Bar diagram shows Smoking (46%)was more common in the patients who had Myocardial Ischaemia and Myocardial Infarction and 8%of the population had the habit of smoking along with alcohol consumption



Craniofacial pain can be the the sole symptom of myocardial ischaemia, failure to recognize its cardiac source can endanger the patient.Patients with acute myocardial infarction who do not experience chest pain run a very high risk of misdiagnosis and death.

One possible source of heterotopic pain is of cardiac origin, which pose a diagnostic challenge. Nearly 6% of the population experience craniofacial pain as the only symptom of cardiac ischemia. Misdiagnosis of these cases may lead to unnecessary dental treatment and a significant number of deaths due to atypical symptoms of coronary disease.

Similarly in the present study 3% of the population experienced craniofacial pain as the sole symptom of Myocardial Ischaemia and Myocardial Infarction.

Prevalence of craniofacial pain in Myocardial Ischaemia and Myocardial Infarction

Craniofacial pain was assessed in both Myocardial Ischaemia and Myocardial Infarction and it was found to be more prevalent in Myocardial Infarction3% as shown below in the table

Table:1

Craniofacial pain	**Myocardial infarction**		**Myocardial ischaemia**		**Total Cardiology cases**	
	No.	**%**	**No.**	**%**	**No.**	**%**
Present	**30**	**3.76**	**4**	**1.97**	**34**	**3.4**
Absent	767	96.24	199	98.03	966	96.6
Total	797	100.0	203	100.0	1000	100.0
Prevalence rate	3.76		1.97		3.4	
'p'	0.147 Not significant					

Fig:20 Out of 1000 patients examined craniofacial pain was prevalent in 34 patients, out of which 30 patients were Myocardial Infarction and four were Myocardial Ischaemia as shown below

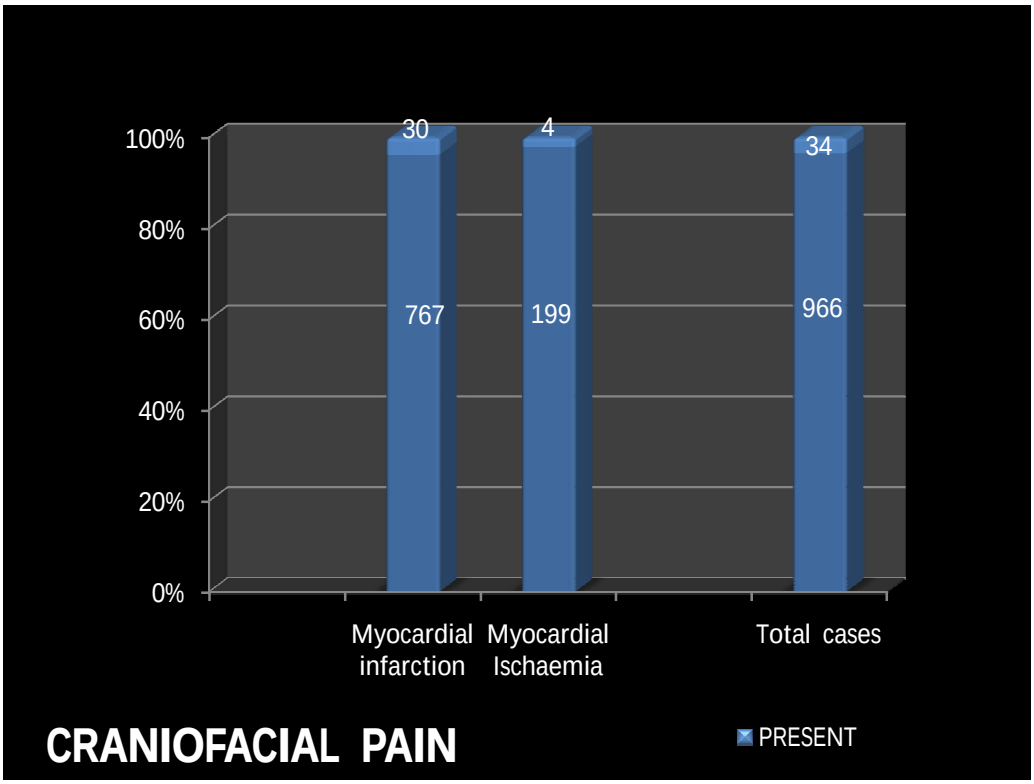


Table:2 **Age and Craniofacial pain**

Craniofacial pain was common in the age group 51-60 with the mean age of 54.41yrs.

Craniofacial pain	**Age (yrs)**	
	Mean	**S.D.**
Present	54.41	9.55
'p'	0.7258 Not significant	

Table:3 **Gender and craniofacial pain**

Gender variation pertaining to craniofacial pain was assessed and it was found that craniofacial pain was more prevalent in females 4% compared with males 3%.

Gender	**Craniofacial pain**			
	Present		**Absent**	
	No	**%**	**No**	**%**
Male (882)	29	3.29	853	96.71
Female (118)	5	4.24	113	95.76
'p'	0.373 Not significant			

Fig:21 depicts gender variation pertaining to craniofacial pain

Endorphins contribute to different pain sensation in women and men. Endorphins rise in ischaemia, are affected by sex hormones, and modulate central pain perception.

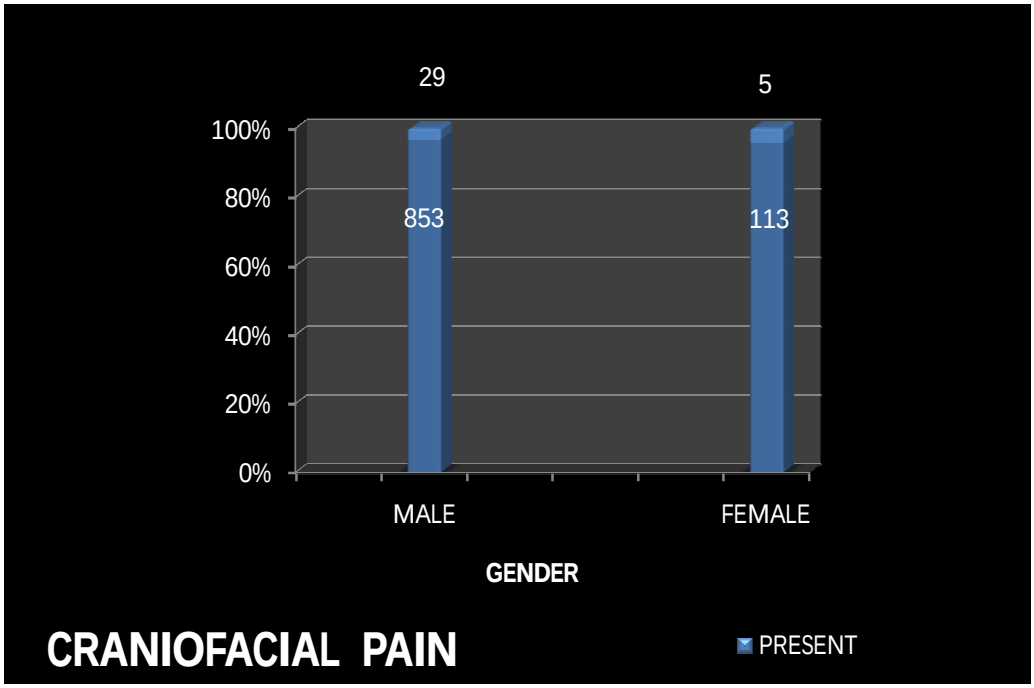


Table:4 **Associated symptoms and Craniofacial pain**

The most common associated symptom experienced by the patients along with craniofacial pain was Profuse sweating followed by dyspnoea and palpitation as depicted below in the table

Associated Symptoms	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Cases with Associated symptoms (447)	17	3.8	430	96.2
Cases without Associated symptoms (553)	17	3.1	536	96.9
Giddiness (34)	1	2.9	33	97.1
Palpitation (34)	3	8.8	31	91.2
Profuse sweating (168)	**7**	4.2	161	95.8
Vomiting & Diarrhoea (49)	1	2.0	48	98.0
Others (5)	-	-	5	100
Dyspnoea (140)	5	3.6	135	96.4
Fainted (17)	-	-	17	100
Total (1000)	34	3.4	966	96.6
'p'	0.6468 Not significant			

Others include: seizures, diarrhoea

Table:5 **Type of craniofacial Pain**

Stabbing type of pain was most common in patients who experienced craniofacial pain as depicted below in the table

Type of pain	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Burning pain (58)	3	5.2	55	94.8
Constricting pain (145)	5	3.4	140	96.6
Stabbing pain (766)	26	3.4	740	96.6
Cases with pain (959)	34	3.5	925	96.5
Cases without pain (41)	-	-	41	100.0
Total	34	3.4	966	96.6
'p'	0.2351 Not significant			

Table:6 **Distribution of Referred pain**

The most common distribution of Referred pain was head ache followed by Right and left shoulder pain.There was no referred pain in Right shoulder,Right and left legs and neck pain

Radiation	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Back pain	3	3.5	82	96.5
Headache	4	10.3	35	89.7
Left shoulder pain	3	2.1	142	97.9
Right shoulder pain	-	-	15	100.0
Right & Left shoulder pain	4	7.4	50	92.6
Neck pain	-	-	12	100.0
Right & Left legs	-	-	7	100.0
Throat pain	1	11.1	8	88.9
Others	-	-	5	100.0
Cases with radiation (374)	15	4.0	359	96.0
Cases without radiation (626)	19	3.0	607	97.0
Total (1000)	34	3.4	966	96.6

Table:7 **Associated comorbidities in the patients who had craniofacial pain**

Hypertensive patients experienced craniofacial pain followed by diabetic patients.

Associated comorbidities	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Cases with Associated comorbidities(196)	11	5.6	185	94.4
Cases without comorbidities(804)	23	2.9	781	97.1
Diabetes (77)	4	5.2	73	94.8
Hypertension (73)	5	6.8	68	93.2
Hypertension & Diabetes (39)	2	5.1	37	94.9
Others (7)	-	-	7	100.0
Total (1000)	34	3.4	966	96.6
'p'	0.0516 Not Significant			

Table:8 Oral status in patients with craniofacial pain

The most common oral presentation was Generalized chronic peridontitis,followed by dental caries

Intraoral examination	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Attrition,cervical abrasion	1	5.6	17	94.4
Partially edentulous	1	4.2	23	95.8
Dental caries	9	2.5	352	97.5
Generalized chronic peridontitis	19	3.8	485	96.2
Smoker's melanosis, Smoker's palate	4	6.9	54	93.1
Total	**34**	**3.4**	**966**	**96.6**

Table:9 **Adverse habits in the patients who had craniofacial pain**.

Smoking was the most common habit in the patients experiencing craniofacial pain

Past history	**Craniofacial pain**			
	Positive		**Negative**	
	No	**%**	**No**	**%**
Cases with Habits	23	3.95	559	96.05
Cases without Habits	11	2.6	407	97.4
Smoking	17	3.7	438	96.3
Smoking & Alcohol	6	7.1	78	92.9
Alcohol	-	-	43	100.0
Total	34	3.4	966	96.6
'p'	0.1689 Not significant			

The causes of referred craniofacial pain (e.g. dental, musculoskeletal, sinusitis etc.) are widely described in the literature and are familiar to clinicians. However, the clinical characteristics of dental and facial referred pain of cardiac origin have not so far been well investigated or compared with pain of dental origin. Because there is a high risk of missed diagnosis when craniofacial pain presents as the sole symptom of myocardial ischaemia and infarction, with consequent risk of fatal AMI, the results of this study should provide clinicians with new data that may be helpful for establishing an appropriate and prompt differential diagnosis.

Pain of cardiac origin where categorized according to age, gender, associated symptoms, location of pain, associated systemic conditions, associated habits, type of pain, distribution of referred pain and oral hygiene status.

- **Age**

In this study on 1000 patients 80% of the population had Myocardial Infarction and 20% had Myocardial Ischaemia and craniofacial pain was more prevalent in myocardial Infarction(3.7%)compared with Myocardial Ischaemia(1.9)% and also Myocardial Infarction was more common between the age groups of 51-60 and the mean age of myocardial Infarction 54yrs similar to study conducted by chaikhouniet al 1983 where the mean age was 51yrs. [20]

According to Muhammed et al 2013 majority of the patients were between the age of 41-70 years whereas in study done by Bahler et al 2015 the mean age of Myocardial Infarction was 68.5 yrs. [20]

Generally risk factors were more strongly associated with acute MI in younger (60 years) compared to older (60 years) women and men.

Interestingly, the protective effects of physical activity and MI and regular alcohol consumption and MI were stronger among older compared to younger men.[20]

- **Gender**

The prevalence of MI was common in males(88%) when compared with females (12%).According to Muhammed et al 2013 majority of the patients with AMI were male (79.8%).According to channama et al 2016 Myocardial Infarction was more prevalent in males with a percentage of 92.5% in males and females 72.5%. [22]

At the turn of century, it was reported that coronary heart disease mortality was expected to increase approximately 29% in women and 48% in men in developed countries between 1990 and 2020. The corresponding estimation in developing countries were 120% in women and 137% in men.[22]

Studies conducted by Hafeez et al and Shabbir et al also showed male dominance . [23]

Albarran et al had also discovered that AMI is more common in males (68%) as compared to females (32%). [24]

Chirsten et al found AMI prevalence was 62% in male.

Risk factors like hypertension and hyperlipidemia are more prominent for men than women in the late 40- to early 50-year range; than their prevalence is higher in women. [22]

Women have an extra protection during their early reproductive life due to the effect of sex hormones. Risk factors which were more strongly associated with MI in women compared to men included hypertension, diabetes, alcohol intake, and physical activity. Only former smoking was more strongly associated with MI in men compared to that in women. [22]

According to Sonia et al 2008 The association of current smoking, consumption of a high-risk diet, abdominal obesity, and psychosocial factors with MI did not vary significantly by sex. [25]

Among women, ApoB/A levels, current smoking, hypertension, and diabetes were more strongly associated with MI in younger compared to older women .Among men, ApoB/A, current and former smoking, hypertension, and abdominal obesity but not diabetes were more strongly associated with MI in younger compared to older men .[25]

According to Hyun Kuk et al Women also experience more silent MIs; nearly half of the MIs which occur in women are unrecognized. Women substantially underestimate their own risk of coronary artery disease and tend to consider their symptoms to other disease processes.One obvious difference between coronary artery disease in men and women is the older age at which it strikes females. [26]

The protection offered by estrogen is believed to play a key role in controlling harmful cholesterol, and maintaining vasodilation of the coronary arteries. Once estrogen production stops, women tend to have reduced effect of this natural protection. By the age 50-55, women start to catch up to men in coronary heart disease rates. By the age 65-70, heart attack rates are similar in both sexes with one in three affected by coronary disease.[26]

- **Associated Symptoms**

a.sweating

In the present study the most common associated symptom was profuse sweating 16.8% of the population in the patients who had Myocardial Ischaemia and Myocardial Infarction which may be due to an increased output of the sympathetic nervous system and 14% of the population had dyspnoea caused by reduced cardiac output leading to left ventricular failure. [27]

According to karnath et al dyspnoea is most commonly associated with anterior infarctions.[27]

b.vomiting and diarrhoea

Heart can no longer adequately pump blood around the body, causing a 'back-up' of deoxygenated blood in the pulmonary circulation , vomiting and diarrhoea occurs due to redirection of blood away from the gastrointestinal system, and increased output of the sympathetic nervous system – particularly due to stimulation of abdominal splanchnic and vagal afferent nerves.

In a study by Arslanian et al 2006 nearly 35% experienced diaphoresis.[28]

And nearly 5% of the population in the present study experienced vomiting and diarrhoea.

- According to Herly et al nearly 55% of the population experienced nausea or vomiting and he also stated that patients with larger infarcts has nausea and vomiting.[29]
- According to Fuller et al nearly 67% of the population experienced nausea and vomiting and he concluded that it was more common with inferior and anterior wall Myocardial Infarction.

Cardiac afferents converge in the spinal cord with input from other somatic organs. Sympathetic afferents converge with somatic input on spinothalamic tract cells and vagal afferents in the nucleus tractus solitarius. The latter may stimulate efferent impulses in the autonomic nervous system, leading to nausea and emesis.[22]

- **Location of pain**

The most common location of pain in the present study was centre of the chest that is precordial chest pain(57%) and 90 % Of the population had pain in right,left,back side of the chest (retro sternal)similar to the study conducted by Muhammed et al 2013, 38% of the population experienced precordial chest pain and in his study 93% of the population experienced pain in and around the chest. Extensive atherosclerosis which reduces blood flow to the heart results in chest pain. Myocardial ischemia can occur as a result of increased myocardial metabolic demand (including extremes of physical exertion and severe hypertension), decreased delivery of oxygen/nutrients (when a thrombus is superimposed on an ulcerated or unstable atherosclerotic plaque) or both .[20]

Occlusion of the coronary artery can activate the cardiac nociceptive afferent fibers . Not only a specific mediator but multiple mediators released at the same time is known responsible for this pain transmission that may interact with one another .

Concentration of bradykinin, thromboxane, adenosine, potassium, histamine and prostaglandins change during MI with bradykinin and adenosine being the most important mediators of cardiac pain and angina pectoris .

Angina-like chest pain stimulated by intravenous injection of adenosine has been reported . Histamine can contribute perception of cardiac pain during MI, through the H1 receptors . Furthermore, bradykinin and thromboxane A2 interact to stimulate cardiac afferent endings that are sensitive to ischaemia and as a result lead to synergistic afferent responses .[6]

There is a significant anatomic overlapping of sympathetic and vagal fibers which are more concentrated in the anterior and posterior walls of the heart, respectively . Sympathetic afferent fibers contribute to most of the pain perception during MI.[6]

However, severing the sympathetic fibers, did not eliminate feeling the pain in the neck and jaws which means vagal fibers are also involved in perception of cardiac pain specially originated from posterior wall of heart .[21]

The cell bodies of the sympathetic and cardiac vagal afferent fibers from the heart are located in the dorsal root ganglia and the ganglion nodosum (nodose ganglion), respectively.[21]

From here the main ascending systems which conduct afferent information to the brain, are the spinothalamic and the spinoreticular tracts.[21]

There are three main sites of infarction anterior,lateral,inferior.Regardles of the site of infarction chest pain is the most common symptom.[21]

According to Kerneth et al chest pain and diaphoresis are the common signs of Myocardial Infarction.

In the present study craniofacial pain was present in 3% of the population.

According to kreiner et al 2014 prevalence of craniofacial pain was present in 12% of the population.There is a wide variation in the prevalence of craniofacial pain between 6 to 30% in several studies.

Levine's sign placing a clenched fist on his sternum to describe his or her chest pain is the classical feature of ischaemic pain.[27]

- **Type of Pain**

The most common description of pain experienced by the patient was stabbing pain 76% of the population. According to Stefan et al Pain character described by the majority in both groups as 'pressure'and burning.[30]

According to Muhammad et al 2008 the most common type of pain experienced by the patients were of constricting type. The most common type of pain in the patients experiencing myocardial infarction were stabbing, constricting, burning which can be used to differentiate the orofacial pain of dental origin.[20]

- **Distribution of Referred Pain**

In the present study nearly 15% of the population experienced left shoulder pain similar to the studies conducted by Muhammad et al 2013.This is because of presence of heart on the left of chest, so pain radiates along left sided cervical nerve roots .[30]

According to Berger et al 1990 chest pain with a wide irradiation involving the right arm strongly suggests that a myocardial infarction is ongoing. Patients may seek chiropractic care due to an insidious onset of neck and upper back pain, secondary to heart problems paint a possible clinical complication.

In the present study nearly 4% of the population experienced pain in craniofacial region along with pain in other regions which suggests that stimulation of the thoracic cardiac branch of the left vagus nerve can produce pain perceived as originating from the left sided throat, neck, jaw and teeth. [31]

- **Associated systemic conditions**

In the present study nearly 8% of the population had diabetes and 7% of the population had hypertension and 4% of the population had diabetes and hypertension. Factors unique to diabetes increase atherosclerotic plaque formation and thrombosis, thereby contributing to myocardial infarction the incidence of CVD mortality was 3-fold higher among individuals with diabetes and non diabetes who had suffered a myocardial infarction .[32]

➢ Role of Diabetes Mellitus in Heart Disease

Diabetes is associated with a relatively greater risk for CVD in women than in men.

Moreover, diabetes negates the normal gender differences in the prevalence of CVD, and when adjusted for other risk factors, the risk rate for increased mortality is 2.4 times greater for diabetic men and 3.5 times greater for diabetic women. [33]

The occurrence of CVD death at the 12-year follow-up was approximately 3 times more in diabetic men than in their nondiabetic controls, regardless of systolic pressure, age, cholesterol, ethnic group, or use of tobacco.[33]

This study also confirmed that systolic hypertension, elevated cholesterol, and cigarette smoking were independent predictors of mortality and that the presence of ≥1 of these risk factors had a greater impact on increasing CVD mortality in persons with diabetes than in those without diabetes.[33]

According to Betsy et al 2008 Moreover, diabetic blood is more likely to be high in triglycerides.[34]

➢ Pathogenesis

Hypertriglyceridemia in diabetes occurs, because insulin promotes the activity of the enzyme lipoprotein lipase, which mediates free fatty acid uptake into adipose tissue (storage) and suppresses the activity of the enzyme hormone-sensitive lipase, resulting in decreased release of free fatty acids into the circulation. [35]

Hypertriglyceridemia can lead to increased production of the small, dense form of LDL and to decreased HDL transport of cholesterol back to the liver.

Dyslipidemia is only one mechanism by which diabetes promotes atherosclerosis; endothelial dysfunction often contributes. Healthy endothelium regulates blood vessel tone, platelet activation, leukocyte adhesion, thrombogenesis, and inflammation.

When these mechanisms are defective, the process of atherosclerosis is accelerated. [35]

Insulin deficiency and insulin resistance promote dyslipidemia accompanied by increased oxidation, glycosylation, and triglyceride enrichment of lipoproteins ,endothelial dysfunction and all of these factors contribute to the increase in atherogenicity, and thus macrovascular disease, found in patients with diabetes.[35]

i.Activated leukocytes

Inflammation is a normal response to tissue injury or pathogen exposure and is a critical factor in the body's ability to heal itself or to fight off infection. The inflammatory response involves the activation of leukocytes (white blood cells) in part, by a family of cytokines and chemokines. Although inflammation is beneficial, if this response is chronically activated it can have a detrimental effect.[35]

Diabetes has long been considered a state of chronic, low-level inflammation, that this immune activation may precede insulin resistance in diabetic and pre-diabetic states and ultimately may be the factor that initially increases cardiovascular risk in these disease processes.[35]

There is a cross-talk between the molecular pathways involved in both inflammation and insulin signalling, provide clues to the strong relationship between insulin-resistant states (such as the metabolic syndrome and type 2 diabetes), inflammation, and CVD.[35]

Proinflammatory cytokines cause or exacerbate injury by a variety of mechanisms including enhanced vascular permeability, programmed cell death (apoptosis), recruitment of invasive leukocytes, and the promotion of reactive oxygen species (ROS) production.

Serum sialic acid can be used as a marker to predict cardiovascular mortality independent of other known risk factors for CVD, including pre-existing CVD.[36]

In addition to diabetes, obesity is associated with increased levels of a number of adipokines (cytokines released from adipose tissue), including tumor necrosis factor-α, interleukin 1β, interleukin 6, and plasminogen activator inhibitor 1 (PAI-1), all linked to the inflammatory response.[36]

The levels of pro-inflammatory cytokines increases as fat mass increases; however, one exception is the adipokine adiponectin, which has anti-inflammatory properties decreases in obese subjects and exacerbates the chronic inflammatory nature of obesity.[37]

Pro-inflammatory cytokines can enhance the production of ROS. The term ROS refers to a subset of molecules called "free radicals." This term refers to any molecule that contains an unpaired electron in the outer orbital. This unpaired electron makes the molecule highly reactive, seeking to either donate an electron to another compound or take up protons from another compound to obtain a stable electron pair. [37]

Because of the reactive propensity of these molecules, ROS can directly damage a number of cell components, such as plasma membranes and organelles.[37]

ROS are produced by the immune system as a way to injure and destroy pathogens, but they are also generated as a result of daily living.[37]

Normal metabolism results in the production of ROS, which act as signalling molecules for both physiological and pathophysiological properties. Oxidative stress occurs when the cellular production of ROS exceeds the capacity of anti-oxidant defenses within cells. [38]

For example, plasma levels of hydro peroxides (one ROS) are higher in subjects with type 2 diabetes compared to nondiabetic subjects, and these levels are inversely correlated with the degree of metabolic control.[35]

The mitochondria are the major source of ROS. At the sub cellular level, the etiologies of insulin resistance and diabetes, as well as their complications, are deeply related to defects in mitochondrial function. The mitochondria produce most of the body's required adenosine triphosphate through the process of oxidative phosphorylation (via the electron transport chain). Oxidative phosphorylation is the major source of ROS under normal physiological conditions, the increased flux of glucose in diabetes has been found to increase ROS production.[35]

Oxidative stress is currently the unifying factor in the development of diabetes complications. According to Brownlee, there are four mechanisms by which chronic hyperglycemia causes diabetes complications:

activation of the polyol pathway; increased formation of advanced glycosylation end products; activation of protein kinase C, an enzyme involved in numerous molecular signalling pathways; and activation of the hexsosamine pathway. [35]

Brownlee and his colleagues found that hyperglycemia-induced mitochondrial ROS production activates each of the four major pathways of hyperglycemic damage. Moreover, blocking ROS production or interfering with ROS signalling attenuated the activity of all four pathways.[35]

Thus, oxidative stress is a crucially important concept in the pathophysiology of the cardiovascular complications in diabetes.[35]

The inflammatory response appears to be over-activated in insulin resistance and in diabetes. Leukocytes are major mediators of inflammation.[35]

They also contribute to the oxidative stress associated with diabetes. ROS are generated not only from the mitochondria, but also from activated leukocytes.[34]

Hokama et al. found that the expression of adhesion proteins on the surface of neutrophils, which suggests activation and ROS production, was significantly increased in diabetes.[34]

Under ischaemic conditions, Hokama et al. found that leukocyte accumulation during reperfusion was enhanced in the diabetic coronary microcirculation, suggesting an increased ability of leukocyte-generated ROS to exacerbate tissue damage after myocardial infarction (MI).[35]

The excess chronic oxidative stress produced in the hyperglycemic state by the mitochondria, as well as the additional acute stress mediated by accumulated leukocytes, may largely explain the mechanism of increased oxidative injury associated with ischemic heart disease in diabetes.

This in turn, aids our understanding of the excessive morbidity and mortality in patients with diabetes after heart attacks when compared to patients without diabetes.[35]

ii. Hypercoagulability

In addition to the alteration in leukocytes in the blood, diabetes is also related to a hypercoagulable state. [35]

The coagulability of the blood is crucially important in ischaemic cardiovascular events because the majority of MI and stroke events are caused by the rupture of atherosclerotic plaque and the resulting occlusion of a major artery by a blood clot (thrombus).[35]

Up to 80% of patients with diabetes die a thrombotic death. Seventy-five percent of these deaths are the result of an MI.[35]

- **Role of Hypertension in Heart Disease**

According to Pedrinelli et al 2012 History of hypertension is a frequent finding in patients with acute myocardial infarction (AMI) and its recurring association with female sex, diabetes, older age, less frequent smoking and more frequent vascular comorbidities composes a risk profile quite distinctive from the normotensive ischaemic counterpart.[39]

Antecedent hypertension associates with higher rates of death and morbid events both during the early and long-term course of AMI, particularly if complicated by left ventricular dysfunction and/or congestive heart failure.[39]

Renin-angiotensin-aldosterone system blockade, through either angiotensin-converting enzyme inhibition, angiotensin II receptor blockade or aldosterone antagonism, exerts particular benefits in that high-risk hypertensive subgroup.[39]

In contrast to the negative implications carried by antecedent hypertension, higher systolic pressure at the onset of chest pain associates with lower mortality within 1 year from coronary occlusion, whereas increased blood pressure recorded after hemodynamic stabilization from the acute ischemic event bears inconsistent relationships with recurring coronary events in the long-term follow-up.[39]

As a matter of fact, excessive diastolic pressure drops may jeopardize coronary perfusion and predispose to new acute coronary events.[39]

According to Francis et al 1983, Both systolic and diastolic hypertension increase the risk of a myocardial infarction and the higher the pressure, the greater the risk .Even when other major risk factors are absent, the increased risk still exists. Almost 40% of patients with ischaemic heart disease who die suddenly have a history of hypertension.[40]

Thus, early in the development of hypertensive vascular disease, cardiac output may be elevated . [40]

However, with advancing vascular disease, progressively increasing total peripheral resistance and developing left ventricular hypertrophy, cardiac output will start to decrease and left ventricular function will deteriorate. In addition, both hypertension and left ventricular hypertrophy have important effects on myocardial perfusion.

Systolic BP Risk Factor MI intraventricular pressure is a key determinant of myocardial oxygen consumption and therefore myocardial oxygen demand will progressively increase as systolic pressure increases. Furthermore, left ventricular hypertrophy is known to be associated with a variety of factors that could adversely affect the response to coronary occlusion. [40]

These include a decrease in density of capillaries , along with failure of vasodilator capacity to increase in parallel with muscle mass , an increase in coronary collateral resistance , more severe subendocardial involvement and larger ischaemic zones . [40]

Thus. myocardial perfusion and ventricular function abnormalities occur in hypertension, independent of those abnormalities known to occur in ischemic heart disease.[40]

- **Role of smoking and Alcohol**

In the present study 46% of the population had history of smoking and 8% of the population had habit of smoking and alcohol and 4% of the population had history of alcohol consumption.

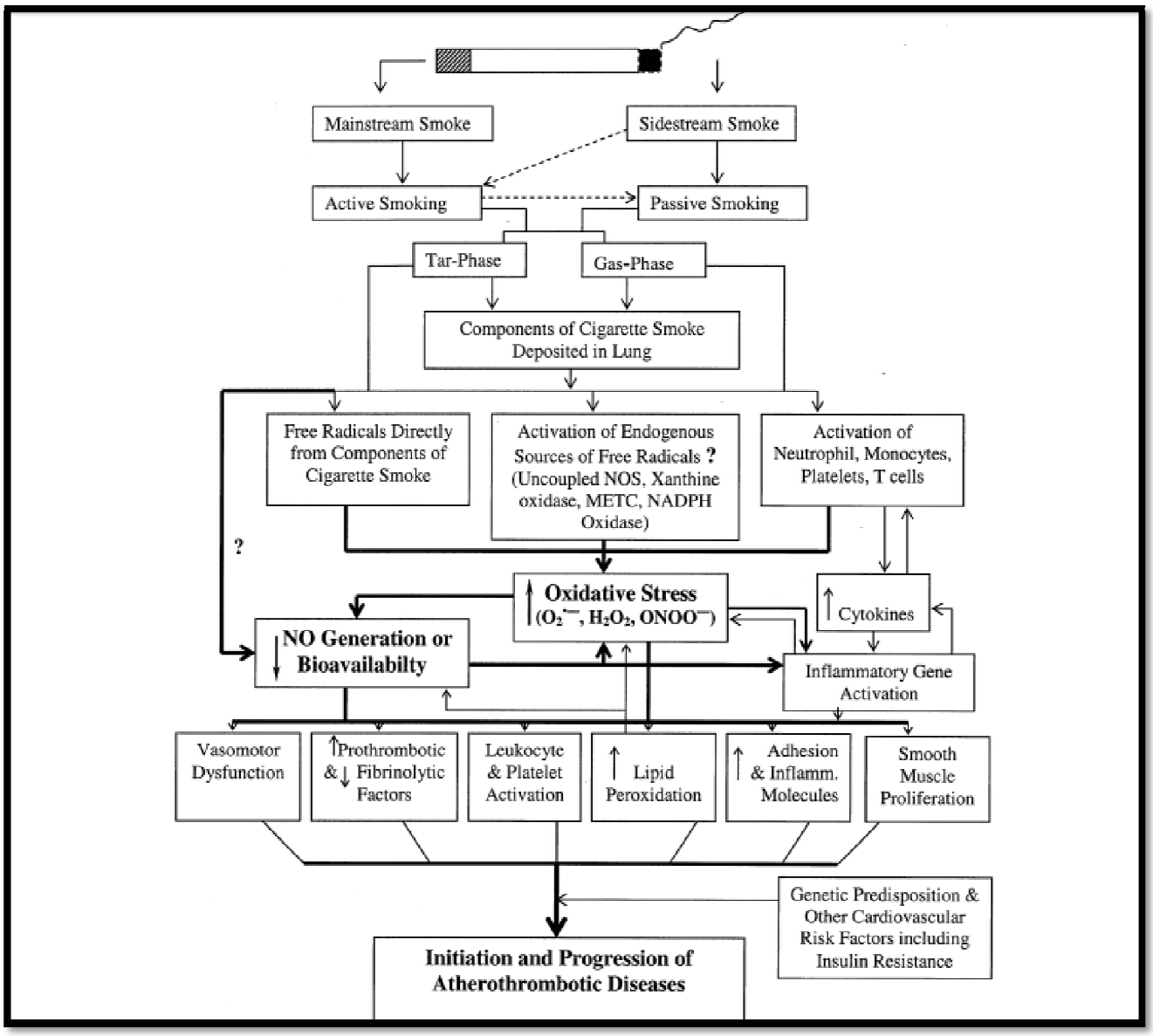


Fig:22 Role of smoking in cardiovascular disease

Courtesy:John A Ambrose The pathophysiology of cigarette smoking and cardiovascular disease J Am coll Cardiol.2004:43(10):1731-1737[41]

According to John et al 2004 Cigarette smoking (CS) continues to be a major health hazard, and it contributes significantly to cardiovascular morbidity and mortality. Cigarette smoking has impact on all phases of atherosclerosis from endothelial dysfunction to acute clinical events, the latter being largely thrombotic. Both active and passive (environmental) cigarette smoke exposure predispose to cardiovascular events. [41]

Cessation of smoking significantly reduces this risk over a one- to three-year period with an exponential decline approaching the risk in ex-smokers within five years of cessation. It was found that there was an immediate reduction in thrombotic events with smoking cessation.[41]

It was found that a citywide smoking ban in public places over a six-month period, reduced the incidence of acute MI by 60% during that time period.[41]

Furthermore, pathologic studies of sudden coronary death indicate that CS increased the risk of plaque rupture and acute thrombosis of a lipid-rich, thin-capped atheroma in men; in female smokers, the prevailing mechanism was plaque erosion with superimposed thrombosis.[41]

Whether there is a distinct direct dose-dependent correlation between cigarette smoke exposure and risk is debatable, it has been shown a non-linear relation to cigarette smoke exposure. The exact toxic components of cigarette smoke and the mechanisms involved in CS-related cardiovascular dysfunction are largely unknown, but CS increases inflammation, thrombosis, and oxidation of low-density lipoprotein cholesterol. [41]

Cigarette smoke exposure increases oxidative stress for initiating cardiovascular dysfunction.[41]

According to Eva Prescott et al 1998ex-smokers reduce their excess risk of myocardial infarction by as much as 50% within the first year after quitting, found that myocardial infarction was more strongly associated with current exposure than with accumulated exposure to tobacco. [42]

This is in agreement with findings that the short term effects of components in tobacco smoke on the haemodynamic system, are more important than the chronic exposure in development of coronary thrombosis.[42]

- **Alcohol as a predisposing factor**

According to Byik et al 2007 Alcohol consumption has both favourable and unfavourable effects on metabolism, lipid profile, blood coagulation and fibrinolysis, blood pressure and vascular tone depending on the amount of alcohol consumed and the way that it is drunk (i.e. drinking habits). It is extremely important to warn people of the risks associated with binge drinking and to encourage them to remain within the recommended safe limits for alcohol consumption.[43]

According to Gowda et al 2003 a case of ST segment elevation myocardial infarction has been reported triggered by heavy alcoholic binge drinking in a man.[44]

Although heavy alcohol use increases the risk of death from cardiovascular complications, there is much evidence suggesting that low to moderate daily consumption of alcohol provides protection against coronary heart disease in both men and women.[44]

- **Oral Hygiene status as a predisposing factor**

In the present study nearly 50% of the population had Generalized chronic peridontitis.

According to Zamirian et al 2008 Advanced peridontitis probably implies a sufficiently long evolution of the disease to become a risk factor for coronary artery disease. [45]

Severe peridontitis has been reported to be associated with a greater thickness of the muscle layer of the coronary artery supporting the role played by the pathogenesis of peridontitis in the formation of atheroma and subsequent acute myocardial infarction.[46]

Advanced peridontitis is characterized by the production of acute episodes of bacteremia that alongside with other factors, can trigger ischemic events.[46]

Destefan, et al. reported a 25% increase in risk for future coronary heart disease in association with PD after adjustment for traditional risk factors.[47]

Joshipura et al. found a significant association between teeth loss due to periodontal disease and CAD in men.[48]

Paunio et al. also reported an association between missing teeth and ischemic heart disease. In addition, edentulous individuals have been shown to have rates of cardiovascular disease equal to or exceeding the rates in individuals with periodontal disease.[49]

- **Craniofacial pain in Myocardial Ischaemia and Myocardial Infarction**

In the present study Craniofacial pain was most prevalent in myocardial Infarction compared with Myocardial Ischaemia. According to kreiner et al 2014 craniofacial pain would be most commonly associated with the areas densely innervated by vagal afferent fibres.[21]

The 5th cranial nerve (trigeminal nerve-TG) is mainly responsible for the majority of pain sensations in the facial area through a very complex neurophysiology and peripheral / central mechanisms . Each of the three branches of TG [including V1 (ophthalmic), the V2 (maxillary) and the V3 (mandibular)] nerves comprise of myelinated and non-myelinated (C) fibers .[21]

Nociceptive input is transmitted via A delta and C fibers that are responsive to neural mediators [Substance P and calcitonin-gene related peptide (CGRP), neuropeptide Y, etc.and are also activated by inflammatory mediators such as bradykinin and prostaglandins. Activation of the free endings of nociceptors involve complex biochemical interactions through membrane receptors or channels including the G-protein-coupled receptor (GPCR), the sodium channels, the voltage-gated potassium channels and the calcium channels .

Once the nociceptors (A delta and C fibers) are activated, the afferent fibers conduct the action potentials to their cell bodies located in the trigeminal (the Gasserian) ganglion.

Then their axons conduct the nociceptive input to the starting point of central nervous system (CNS) (brainstem) and the trigeminal spinal nucleus (TSN) .[21]

The most caudal zone of TSN is called subnucleus caudalis (located in the medullar dorsal horn) where the primary nociceptive afferent neurons of TG synapse with the second order neurons.[21]

The involved neurotransmitters in this synapse are glutamate, substance P and CGRP along with several modulatory circuits. [21]

From the sub-nucleus caudalis, the second order (projection) neurons project the input to several sites within the central nervous system, including the thalamus that act as a relay for ascending nociceptive inputs to the sensory cortex. [21]

Convergence mechanisms are involved in craniofacial pain, which originates in the heart. The upper cervical spine segments are convergence areas for trigeminal, visceral and phrenic inputs. The trigeminal sub-nucleus caudalis, receives extensive convergence inputs from cutaneous, muscular and visceral afferents .[21]

There is a connection between cardiac vagal afferents and trigeminal and trigemino-thalamic neurons; moreover, cardiac inputs have a modulatory action on the trigeminal system .[21]

According to Qin et al , 89% of the C1-C3 neurons that receive somatic inputs from the craniofacial structures, are also excited by cardiac nociceptive afferent fibers which can justify the referred pain to the face during MI. [21]

In other words, C1-C2 spinal neurons may act as an integrating centre for cardiac nociceptive from both vagal and sympathetic afferent fibers . [21]

However, vagal afferents from the heart contribute to craniofacial pain more commonly because somatic fields in the jaws and the neck were are shown to be more reactive to vagal than to sympathetic experimental electrical stimulation.[21]

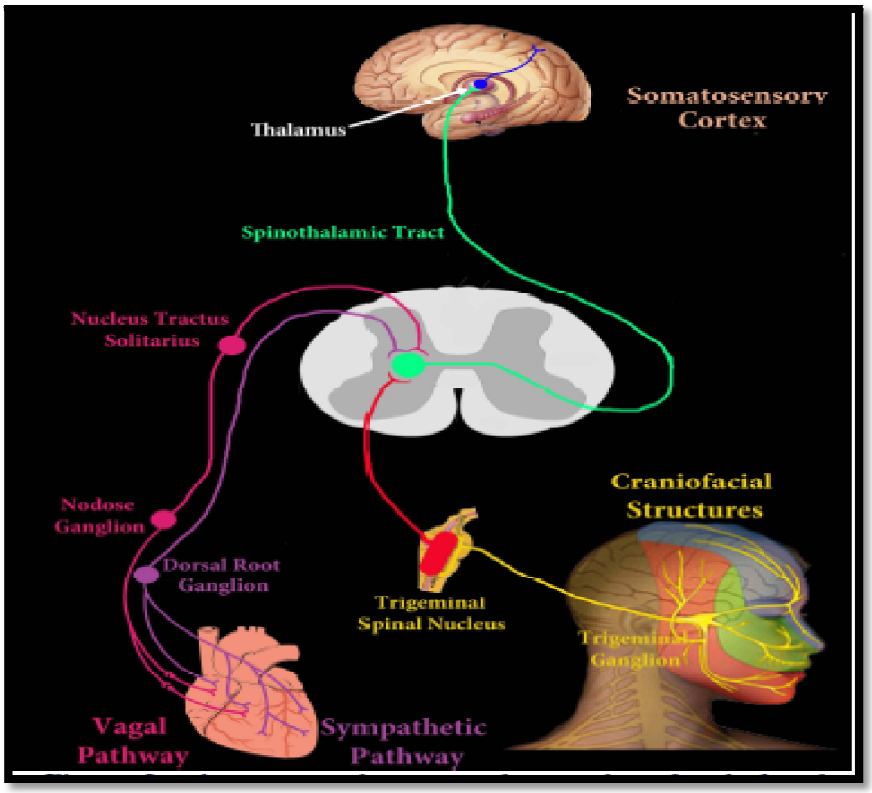


Fig:23 Depicts the craniofacial pain pathway in Myocardial Ischaemia and Myocardial Infarction

Courtesy:. Mahta Fazlyab,et al. Craniofacial Pain as the Sole Sign of Prodromal Angina and Acute Coronary Syndrome: A Review and Report of a Rare Case. Iran Endod J. 2015 Fall; 10(4): 274–280.[21]

Pain of cardiac origin and manifesting in the orofacial area may irradiate to other craniofacial structures (throat, neck, temporal area, head, infraorbital region, maxilla) or to the thorax region (thorax, shoulders, arms). Odontogenic pain (pulpal or periodontal) can be reflected in structures such as the ears and the temporal area. [6]

According to Kreiner et al. 32% of the patients presented concomitant craniofacial pain in other regions and only 6% presented craniofacial pain as the only symptom during the ischaemic episode.[51]

In the present study prevalence of craniofacial pain was more in females compared with males. Gender differences regarding symptom presentation during an AMI were reported by several studies, with women being less likely to report discomfort in the centre of the chest (Kannel, 2002; King and McGuire, 2007) and more likely to report digestive symptoms, palpitations, nausea and unusual fatigue (DeVon et al., 2008). However, one study did not find gender differences when comparing typical and atypical symptoms
during an AMI (Isaksson, 2008).Danesh et al 2012 reported that craniofacial pain was more common in females.[19]

According to Martha et al 2011 Throat, jaw, and neck discomfort were reported more by women, which is consistent with many previous studies. The observation that women have greater vagal activity than men, and that the jaw area is innervated by the vagus nerve may be clues to the mechanism underlying this observation.[50]

According to Deborah et al 1997 retrospective analyses of AMI patients have suggested that women have “atypical presentations” (non–chest pain) more often than men. According to Myer 2008 Other than anterior chest pain, women may experience pain in locations such as the lower jaw and teeth, both arms, shoulders, neck, upper back and epigastrium.[51]

Atypical presentation in ACS was observed more commonly in women than men in large cohort studies . Women with coronary heart disease are older by 10 years and have more risk factors than men. It might be due to lack of early recognition and management. There are several differences between men and women in presentation. [52]

Women were less likely to have typical angina, rated their pain as more intense, used different words to describe it (more burning, sharp), and reported more non-pain-related symptoms than men. They experienced pain and other sensations in the neck area more frequently. Another feature of chest pain in women is that angina being induced by rest, sleep, mental stress instead of or addition to physical exertion. Psychosocial factors might also affect symptom presentation and diagnostic approach in women. [53]

As women underestimate their own risk of coronary artery disease, diagnostic approach by physician could be altered less aggressively than men. [53]

Compared with men, women are less likely to perform cardiac monitoring, cardiac enzyme measurement, electrocardiogram, cardiac consultation, admission to a coronary care unit, undergo less coronary angiography, angioplasty, and bypass surgery.[54]

According to vera et al Pain generation in the heart is a complex process. It starts with the stimulation of afferent nerve endings in the heart. [55]Nerve growth factor (NGF) is one of the substances involved in cardiac pain sensation. NGF leads to vallinoid receptor activation and stimulation of afferent nerves in the heart. It has not yet been established whether vallinoid receptors and NGF are differently expressed in female and male hearts.[22]

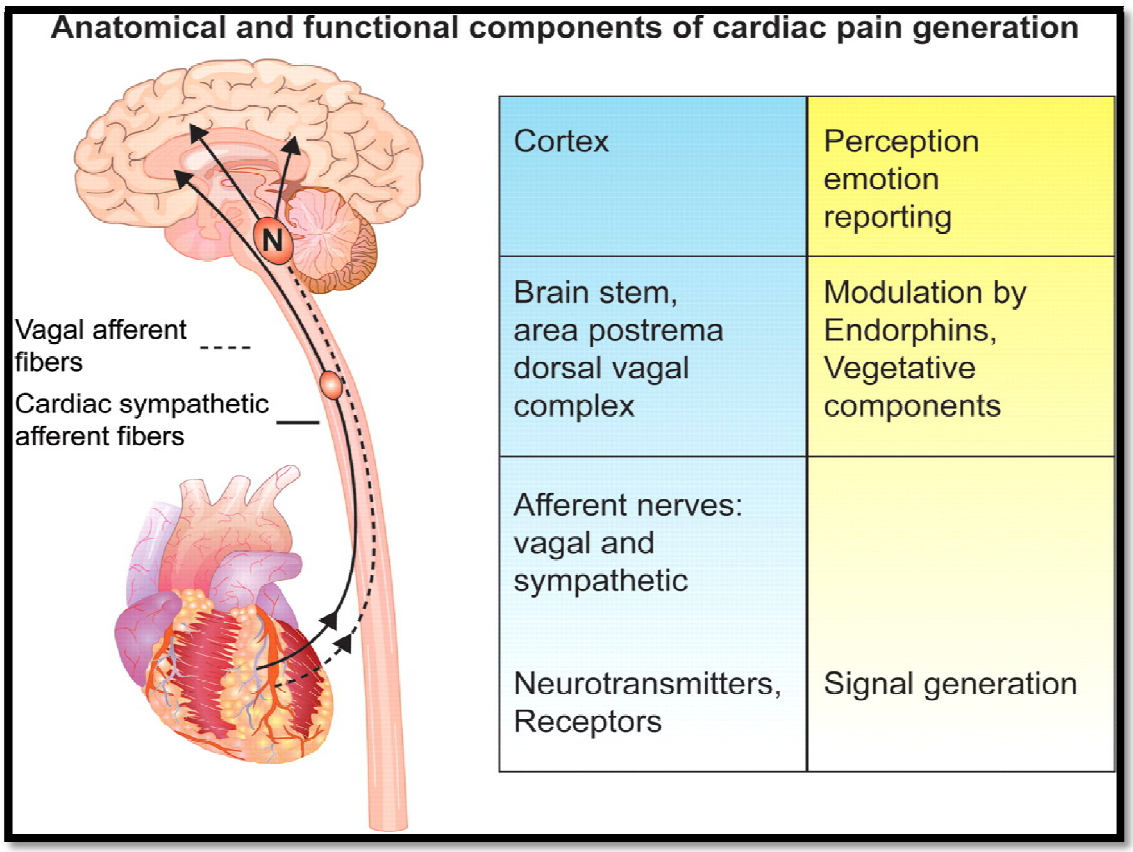


Fig:24 components involved in pain generation of cardiac origin

Courtesy: Vera Regitz-Zagrosek. Sex and gender differences in symptoms of myocardial ischaemia 2011;32(24): 3064-3066[22]

Transmission of pain occurs by activation of sympathetic and vagal nerves. Sympathetic nerves predominate on the anterior surface of the heart where perfusion mainly comes from the left anterior coronary artery, whereas parasympathetic nerves arise from the posterior and inferior surfaces, mainly perfused by the right coronary artery or left circumflex artery. Thus, activation of the parasympathetic nervous system, which is more related to nausea and emesis, may characterize pain in patients with right dominant systems. [22]

This may also give rise to sex differences since right dominant coronary systems have been reported to be more frequent in women.[22]

Endorphins may also contribute to different pain sensation in women and men. Endorphins rise in ischaemia, are affected by sex hormones, and modulate central pain perception. [22]

In the present study patients with hypertension(7%) had craniofacial pain when compared with diabetic patients(5%) . According to Kreiner et al 2014 Craniofacial pain was more prevalent in Diabetic patients.

The symptomatology of myocardial Ischaemia and Infarction , which comprises both pain and non-pain symptoms, may be affected by traditional risk factors such as age, gender, smoking, hypertension, diabetes and metabolic syndrome.[53]

As diabetes mellitus progresses, it results in endothelial dysfunction and changes in energy metabolism which lead to atherosclerosis in medium- and large-caliber arteries, creating lesions in coronary, cerebrovascular and peripheral arteries. Additionally, atherosclerotic plaques tend to develop much earlier, advance more swiftly and are more diffuse in diabetic patients than in non-diabetics. These factors contribute to a two to four-fold higher risk of cardiovascular events in diabetics compared to non-diabetics, with cardiovascular disease being the main cause of death.[53]

Nerve growth factor and vallinoid receptor activation are reduced in diabetic hearts, and this may contribute to the occurrence of silent ischaemia in patients with diabetes.[53]

Neuropathy is particularly frequent in diabetic patients, the elderly, and smokers, and may be mediated by RAGE and the NF-κB pathway.[53]

Diabetic neuropathy is also related to reduced synthesis of NGF, and depletion of calcitonin gene-related peptide (a marker for nociceptory nerves)-containing neurons.[53]

Autonomic neuropathy may predispose to infarction and result in atypical presenting symptoms in the diabetic patient, making diagnosis difficult and delaying treatment.

The clinical course of myocardial infarction is frequently complicated and carries a higher mortality rate in the diabetic than in the nondiabetic patient.[36]

One study reported that younger individuals with diabetes run a 2.5 old higher risk in experiencing atypical symptoms than individuals with non diabetes.[36]

In the present study diaphoresis was present in 4% of the population along with craniofacial pain. According to Muhammed et al 2008 persons who experienced craniofacial pain also experienced profuse sweating as one of their symptoms similar to the present study.[28]

In the present study Pain type was of stabbing in 76% of the population, stabbing was also the type of pain 3.4% described by the patient who experienced craniofacial pain . The quality of craniofacial pain from cardiac or dental origin is different, implying a high diagnostic validity.[28]

According to Kreiner et al 2014 the most common description of pain were pressure and burning when describing the facial pain of cardiac origin.

According to Mahta et al 2015 Patients describe referred cardiac pain as a tight and burning .

In the present study the most common radiation of pain is left shoulder15% but in the patients who had craniofacial pain the most common radiation of pain was in throat 11%.

Smoking was most common in 46% of the population whereas patients with craniofacial pain had history of smoking as well as consumption of alcohol in 7% of the population.

In the present study patients who had history of smoking and alcohol consumption experienced craniofacial pain.

According to Muhammad et al 2008,Smokers had craniofacial pain compared with others.

Neuropathy is particularly frequent in diabetic patients, the elderly, and smokers, and may be mediated by RAGE and the NF-κB pathway thereby atypical chest pain is most commonly experienced by smokers.[22]

Intraoral Examination was done to rule out whether the craniofacial pain is due to dental origin and 50% of the population had generalized chronic periodontitis .It is associated

with the incidence of coronary heart disease (CHD) among younger men, independent of established cardiovascular risk factors.

Cumulative evidence supports a causal association between periodontal infection and artherosclerotic cardiovascular disease or its sequelae. [56-58]

The possible link may involve direct and indirect effects of the periodontal infection; an alternative pathway may be related to genetic and other host factors that increase the susceptibility to both atherosclerosis/thrombosis and chronic peridontitis .[56]

Studies have shown that peridontitis results in higher systemic levels of C-reactive protein, interleukin (IL)-6, and neutrophils. These elevated inflammatory factors may increase inflammatory activity in atherosclerotic lesions, potentially increasing the risk for cardiac or cerebrovascular events.[59-60]

These systemic markers of inflammation are also said to serve as predictors of present and future cardiovascular events and disease.[59]

In addition, oral bacteria have been found in carotid atheromas and it is reported that some oral bacteria may be associated with platelet aggregation, an event important for thrombosis.

Evidence suggests strong association between chronic oral infections and myocardial infarction .[60]

Craniofacial pain of cardiac origin was the sole symptom in 3.4% of patients. However, in the absence of chest pain, craniofacial structures were affected. The association of pain with associated symptoms could be helpful to suspect craniofacial pain of cardiac origin during history taking.

The following conclusions were made from the study

- ✓ Craniofacial pain was more prevalent in Myocardial Infarction
- ✓ Craniofacial pain was more prevalent in the age group of 51-60
- ✓ Craniofacial pain was more prevalent in females
- ✓ Craniofacial was more prevalent in hypertensive patients
- ✓ The most common pain descriptor in craniofacial pain of cardiac origin was stabbing
- ✓ Craniofacial pain was most commonly associated with profuse sweating
- ✓ Craniofacial pain was more prevalent in the patient who had habit of smoking
- ✓ The most common oral manifestations in patients of Myocardial Infarction and Myocardial Ischaemia were Generalized chronic peridontitis

Awareness of the characteristics of craniofacial pain of cardiac origin is necessary for early diagnosis. Cardiac induced referred pain to the craniofacial region may drive a referral to oral physicians. To initiate prompt and appropriate treatment, oral physicians and medical clinicians should be made aware of the clinical characteristics of craniofacial pain of cardiac origin. Oral physician should ensure appropriate emergency care before further referral. So Once pain of cardiac origin is identified , insufficient oxygenation to the heart should be prevented . Therefore, the management of all medical emergencies should ensure that oxygenated blood is being delivered to these critical organs. Oxygen is indicated for every emergency except hyper-ventilation. This should be done with a clear full face mask for the spontaneously breathing patient and a bag-valve-mask device for the apneic patient. Therefore whenever possible, with the exception of the patient who is hyperventilating, oxygen should be administered.

This provides the skills to manage most medical emergencies, which begin with the assessment of vital signs , and if necessary the treatment of airway, breathing and circulation (the ABCs of CPR). Usually, only after these ABCs are addressed the dentist should consider the use of emergency drugs.

Nitrogycerine drug is available as sublingual tablets or a sublingual spray indicated for acute angina or myocardial infarction. It is characterized by a rapid onset of action.

Aspirin (acetylsalicylic acid) 162 mg is one of the recognized life-saving drugs should be given immediately to any patient with pain suggestive of acute myocardial infarction, as it has been shown to reduce overall mortality from acute myocardial infarction.

The purpose of its administration during an acute myocardial infarction is to prevent the progression from cardiac ischaemia to injury to infarction. There is a brief period of time early on during a myocardial infarction where aspirin can show this benefit.

“Prompt and earlier the diagnosis better the prognosis.” To conclude craniofacial pain is multifactorial ,but cardiac origin should be considered when pain is burning, stabbing in nature radiating bilaterally to the jaws associated with symptoms such as dyspnoea , diaphoresis ,palpitations and giddiness. This needs to be considered as cardiac emergency.

Cardiovascular disease is the leading cause of mortality world wide the associated morbidity affects all walks of life and has a great impact in the quality of life of affected individuals. Hence when oral physician encounters craniofacial pain with associated symptoms, cardiac origin should be considered. so early recognition and prompt referral can prevent morbidity and mortality rates.

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ANNEXURE I

Ref:UT:BDSC:IRB-EC/2014 Date:18.11.2014

From

Institutional Review Board-Ethical committee,
Best dental science college,
Madurai .

To

The Controller of Examinations ,
The Tamil Nadu DR.MGR Medical University,
No. 69, Anna salai,
Guindy,
Chennai-600 032

Sir/Madam

The Dissertation topic titled " PREVALENCE OF CRANIOFACIAL PAIN AS ONE OF THE SYMPTOMS OF MYOCARDIAL ISCHAEMIA AND MYOCARDIAL INFARCTION" submitted by DR.A.STEFFINA LYDIA JASCINTH postgraduate student has been approved by Institutional Review Board of Best Dental Science College on 18.11.2014.

DR.K.S PREM KUMAR.M.D.S.,
VICE PRINCIPAL
MEMBER SECRETARY
INSTITUITONAL REVIEW BOARD-ETHICAL COMMITTEE
BEST DENTAL SCIENCE COLLEGE
MADURAI

DR.PURUSHOTHAM MANVI.,M.D.S.,
PRINCIPAL
BEST DENTAL SCIENCE COLLEGE
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ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate	:	Dr.A.STEFFINA LYDIA JASCINTH
Course	:	PG in M.D.S. DENTAL
Period of Study	:	2014 -2017
College	:	**ULTRA'S BEST DENTAL SCIENCE COLLEGE**
Research Topic	:	**PREVALENCE OF CRANIOFACIAL PAIN AS ONE OF THE SYMPTOMS OF MYOCARDIAL ISCHAEMIA AND MYOCARDIAL INFARCTION**

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

R. Panneerselvam 29/04/2015

Member Secretary

Dean/Convenor

29 APR 2015

INFORMED CONSENT

Participant information sheet

Title of Research: Prevalence of Craniofacial pain as one of the symptoms of Myocardial Ischaemia and Myocardial Infarction

Introduction of the proposed Research:

Orofacial pain of cardiac origin needs to be identified since the success of treatment depends on identifying the source of pain rather than locating the site of pain thereby providing correct treatment for cardiac patients. You are being asked to participate in this research study .You will be asked about the history, nature of pain , duration of pain,associated symptoms and comorbidities and Habit history. However , before you give your consent to take part in this study , you must read and understand the explanation of the proposed study . It describes the purpose and benefits of the study . After you have read it , please feel free to ask any questions you may have . This is to make sure you understand , what your participation in this study may involve , before you sign and date the consent form. Your participation in this study is voluntary . The results of your samples will be kept confidential and used for the research only.

Informed Consent

I confirm that I have read the participant information sheet on ____/____/____ it has been read to me, I understand it, I have had the opportunity to ask questions about it, and that my questions have been answered to my satisfaction.

I have no objection in being a part of this study. The operator has promised to keep the confidentiality of my personal records. I understand that my participation is voluntary and I am free to withdraw at any time.

(In case of illiterate participant the information is explained and thumb impression is obtained in the presence of an unrelated witness.)

Name of the Participant: _____

Signature or thumb impression: _____

Date: _____

ANNEXURE II

Informed Consent

I confirm that I have read the participant information sheet on 1/6/2015 it has been read to me, I understand it, I have had the opportunity to ask questions about it, and that my questions have been answered to my satisfaction.

I have no objection in being a part of this study. The operator has promised to keep the confidentiality of my personal records. I understand that my participation is voluntary and I am free to withdraw at any time.

(In case of illiterate participant the information is explained and thumb impression is obtained in the presence of an unrelated witness.)

Name of the Participant: Muniyandi

Signature or thumb impression:

Date: 1/6/2015

ANNEXURE II

ஆராய்ச்சி ஒப்புதல் படிவம்

ஆராய்ச்சியின் தலைப்பு : கிரானியோ-பேசியல் வலி நோய் வந்தால் இதயத்தில் குருதியோட்டம் மற்றும் மாரடைப்பின் அறிகுறிகள் குறித்த ஆய்வு

பெயர் : தேதி :

வயது : ஆராய்ச்சி சேர்க்கை எண் :

பால் : ஆண் / பெண்

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விவரங்களை நான் புரிந்துகொண்டு எனது முழு மனதுடன் சம்மதிக்கிறேன். இந்த ஆராய்ச்சியின் போது வாய் பரிசோதனை செய்து கொள்ளவும், நான் சம்மதிக்கிறேன். இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியில் இருந்து எந்த நேரமும் பின்வாங்கலாம் என்றும் அதனால் எந்த பாதிப்பும் எனக்கு ஏற்படாது என்பதையும் புரிந்து கொண்டேன். நான் எனது சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் பங்குபெற சம்மதிக்கிறேன். இது சம்மந்தப்பட்ட ஆய்வு அறிக்கையை மேற்குறிப்பிட்ட நபரோ அல்லது அமைப்போ பயன்படுத்த நான் மனப்பூர்வமாக சம்மதிக்கிறேன்.

பங்கேற்பாளரின் கையொப்பம்:.....தேதி :.....

ANNEXURE III

Case History format

O.PNO:

Name:

Age:

Sex:

Address:

Medical History:

Medical line of treatment for MI:

Investigations subjected for MI:

Associated Symptoms:

Pain location:

Type of pain:

Duration of pain:

Irradiation:

Intensity:

Frequency:

Aggravating factors:

Relieving factors:

Past Dental History:

Whether He had tooth pain ,jaw pain before angina?

Whether the pain occurred unilaterally or bilaterally?

Did he visit the dentist?

How many days before the onset of angina he visited dental clinic?

Case History format

What treatment has been done?

Was the tooth pain,jaw pain relieved after dental treatment?

Personal History:

General Examination:

Built :

Cyanosis: Clubbing: Oedema:

Icterus: Organomegaly(if any)

Nails:

Vital Signs:

Temp:

Pulse rate:

Respiratory rate:

Blood pressure:

INTRA ORAL EXAMINATION

Treatment done:

Was the dental pain relieved after cardiac treatment?

If persists tooth involved:

Reason for pain:

ANNEXURE IV

Sl. No	Name	Age	Sex	Past medical History	Associated Symptoms	Pain Location	Type of Pain	Radiation	Intraoral examination	Personal History	Investigations done at Cardiac unit	Diagnosis in the cardiac unit
1	sivanandi	60	Male		Profuse sweating	centre of chest	stabbing pain		Smoker's melanosis , Smoker's Palate	Smoking and Alcohol	ECG:ST Elevation	myocardial infarction
2	anandaraj	51	Male		Profuse sweating	left side of chest	burning sensation		Smoker's melanosis , Smoker's Palate	Smoking	ECG:ST Elevation	myocardial infarction
3	Muniyandi	66	Male		Dyspnoea	centre of chest	stabbing pain		Dental Caries	Smoking	ECG:ST Elevation	myocardial infarction
4	Pandi	43	Male			centre of chest	burning sensation		Dental Caries	Smoking	ECG:ST Elevation	myocardial infarction
5	Kannan	65	Male		giddiness	centre of chest	stabbing pain	neck pain	completely edentulous	No	ECG:ST Depression	myocardial Ischaemia
6	govindaraj	55	Male		Dyspnoea	left side of chest	burning sensation		Dental Caries	No	ECG:ST Elevation	myocardial infarction
7	ramasamy	63	Male	Diabetes	Dyspnoea	left side of chest	stabbing pain		Generalized chronic peridontitis	Smoking	ECG:ST Depression	myocardial Ischaemia
8	sikkanthar	63	Male	Hypertension and Diabetes	Dyspnoea	centre of chest	stabbing pain		Generalized chronic peridontitis	Smoking	ECG:ST Elevation	myocardial infarction
9	karunanidhi	53	Male		Profuse sweating	centre of chest	stabbing pain		Generalized chronic peridontitis	Alcohol	ECG:ST Depression	myocardial Ischaemia
10	karuppaya	48	Male	Hypertension and Diabetes	vomiting and diarrhoea	left side of chest	stabbing pain		Generalized chronic peridontitis	No	ECG:ST Elevation	myocardial infarction

……Continued